THE
BRITISH JOURNAL OF SURGERY

VOL. XXIV JULY, 1936 No. 93

IPSISSIMA VERBA

BY SIR D'ARCY POWER, K.B.E., LONDON

X. A CASE OF ACUTE PERFORATIVE APPENDICITIS

As this number of the BRITISH JOURNAL OF SURGERY is issued to commemorate the good service Lord Moynihan has rendered it for more than twenty years, it may not be inappropriate to recall one of the earliest, if not the very earliest, of his publications. He read the case at a meeting of the Leeds and West Riding Medical Society in May, 1895, when he was aged 30, and was an Assistant Surgeon at the Leeds General Infirmary. It is entitled, "A Case of Acute Perforative Appendicitis followed by Septic Peritonitis; Abdominal Section: Recovery". The communication afterwards appeared in *The Lancet* (1896, ii, p. 1806), and is here slightly shortened. It runs:—

"Cases of recovery after abdominal section performed for acute septic peritonitis depending on disease of the vermiform appendix are sufficiently rare to warrant the publication of any that may occur. In the early part of last year I was fortunate enough to have such a case. The patient, aged 13, an exceptionally bright and intelligent boy, suffered two years ago from some illness of one week's duration, characterized by abdominal pain, constipation, and a general feeling of languor and sickness without actual vomiting. From this date until March 29th he was perfectly well, but on that day he had in the early morning a loose stool with some abdominal discomfort hardly amounting to pain. The pain continued to increase until on April 1st it had become serious and was accompanied by abdominal distension. Examination showed that the pulse was rapid and feeble, the temperature 100·6° F., the skin clammy and the abdomen uniformly distended and everywhere tender but without any localized resistance. An operation was decided upon, the general peritoneal cavity being first opened by an incision through the linea alba and then cleansed thoroughly, the cæcal region being afterwards exposed through a separate incision and dealing with whatever was found there.

"On opening the peritoneal cavity a large quantity of curdy looking, highly offensive pus made its escape. On introducing my finger into the abdomen I was able to make out that there was a moderate sized collection of pus in Douglas' pouch and I could feel an opening distinct though small which led to an abscess cavity in the neighbourhood of the appendix in the iliac fossa. I then thoroughly cleansed the whole peritoneal cavity, spending some time over it and taking pains

to ensure that no small collection of pus hemmed in by adherent coils of intestine, escaped notice. I then put a plug of iodoform gauze into the cæcal region through the opening I had found and incising the wall above Poupart's ligament I exposed the abscess cavity. Its lining was shreddy, irregular, and in places almost gangrenous and though I have no doubt that the appendix was the cause of the formation of the abscess I was unable to identify it unmistakably and therefore could not remove it. I cleansed the abscess as perfectly as I could by scraping

LORD MOYNIHAN, aged 30

gently, sponging firmly with iodoform gauze and flushing with boiled water. I then dusted the whole cavity with a little iodoform and employed a glass tube to drain the general peritoneal cavity and a stout indiarubber tube to drain the cæcal region.

"The patient rallied after the operation much better than we either hoped or expected. He remained in bed for nine weeks and though in danger for perhaps the first sixty hours, there was no doubt after that time that the ultimate issue of the case would be satisfactory. The boy is now quite recovered and as healthy and vigorous as ever.

" The class of case similar to the one I have just described is of course almost invariably fatal. During the whole of the time that I was house surgeon and resident surgical officer at the [Leeds] Infirmary there was not a single case of recovery from this condition and until the last two or three years not a single recovery had, so far as I know, been put on record and even up to the present time the successful cases in England number only units. But that recovery should be, and will eventually be, the rule instead of the exception I feel complete confidence. In pondering over the matter I have thought that it might be possible to formulate certain essentials on the strict observance of which I feel complete confidence. They are (1) Make an early diagnosis; (2) If at the end of say twenty-four hours the symptoms are not retrogressing operate at once; (3) If the case is first seen when the abscess has formed do not be content with merely opening and draining the cavity, but, if possible remove the appendix as well; (4) If the case is not seen until an early diagnosis of perforation has been made operate immediately, cleansing thoroughly but avoiding, if possible, lavage of the peritoneum. If these four rules for treatment be efficiently carried out I believe that a larger percentage of cases will recover than ever before and that every patient who has recovered will be exempt from any similar attacks in the future."

It should be remembered that Moynihan gave this advice in 1895, when inflammation of the appendix was just coming into notice. The number of operations at St. Bartholomew's Hospital in that year was 10, and 3 of the patients died. The general surgeons still feared the peritoneum, and there was a distinct cleavage of surgical opinion as to the treatment of a condition with which they were as yet unfamiliar. The older school advised waiting until the acute symptoms had passed away. They operated when there was an abscess or after a delay of three weeks. The younger generation advocated immediate operation, and to this school Moynihan belonged.

I am indebted to Lady Moynihan for the photograph, which she believes was taken in or about the year 1895.

LORD MOYNIHAN
A PERSONAL APPRECIATION

By RUTHERFORD MORISON, Newcastle-upon-Tyne

It is almost fifty years since I first met Moynihan. I was then in practice at Hartlepool, and had operated upon a patient in whom he was interested. He wrote asking if I could see him and tell him all about the case if he came through—and he did. I suspect, too, that he wanted to see what sort of fellow I was.

He was then tall, thin, and very pale, but so alert that he would now be described as a "real live wire"; but with complete control. Nothing escaped his notice, and he wanted to know more than I could tell him about everything. At lunch we were honoured, as I always was, by the company of an enormous castrated tom cat, and afterwards we walked to the hospital to visit some malaria patients under my care. On my eightieth birthday, two years ago, he wrote the kindest letter, and asked if I remembered the cat and the malaria. Of course I did, and I also recall that as we walked to the hospital he said he did not think the majority of surgeons knew enough anatomy—they should at least know the landmarks of the epiphyses and remember when their fusion with the shafts occurred.

I remember so clearly our conversation on this first meeting because at this time I was mentally starved, so far as my professional life was concerned. My colleagues in Hartlepool were old and had long given up any idea of inquiring into the why and wherefore of their work. The delight of meeting and talking with an enthusiast such as Moynihan can be readily imagined. We did not then, and have not since, always seen eye to eye on all the problems we tackled; but our differences of opinion have but added to the zest and enjoyment of our long friendship.

Hartlepool, when Moynihan visited it, was a very old seaport and fishing town between the Tees and the Tyne on the east coast, sleepy but holding its position chiefly as an importer of timber and exporter of coal.

The next time I saw Moynihan was at Leeds Infirmary, where I had gone to see Mayo Robson operate. Moynihan was Resident Surgical Officer, and his chief interest at that time was in fractures. From this time I noticed with interest Moynihan's steady and inevitable rise to the highest attainable positions, gained by the sheer force of such irresistible ability as could not be ignored.

In the following years I frequently met him. About fifteen years ago I visited Leeds and saw him perform two operations. The first patient was a thin fragile old lady with a long history of stomach trouble, pain, frequent vomiting, and a lump at her pylorus, the result of chronic ulceration of the lesser curvature involving the pylorus. Half of her stomach was excised. The second patient was a fat middle-aged woman sent in as an abdominal emergency. She had been suddenly seized by overwhelming pain and vomiting, and had developed a high temperature. Her swollen gall-bladder could be felt, and an acutely inflamed, tense, and nearly gangrenous gall-bladder was excised—a typical example of those cases where a stone blocks the neck of the gall-bladder when it has just started to empty itself and the

septic bile cannot escape. Acute infection and tension complete the destruction.

Both of these operations thrilled me, because they showed such perfect art. What is the secret of such success? Surely that the surgeon has learned to make his operation so easy that any hitch was incredible. We have all heard how *easy* golf is to Bobby Jones, and, for the same reason, I do not doubt it.

Though Lord Moynihan's skilful and beautiful operations will not soon be forgotten, my feeling is that the future will attach more importance to his founding of the Moynihan Chirurgical Club. He has drawn together a group of the rising younger surgeons to educate themselves by meeting twice a year: once to spend a week together in hospitals abroad, and the second time in a daily visit to a hospital in which one of them works. It is difficult to over-estimate the value of this experience, not only to the surgeons and their patients, but to the whole community.

(R. M.)

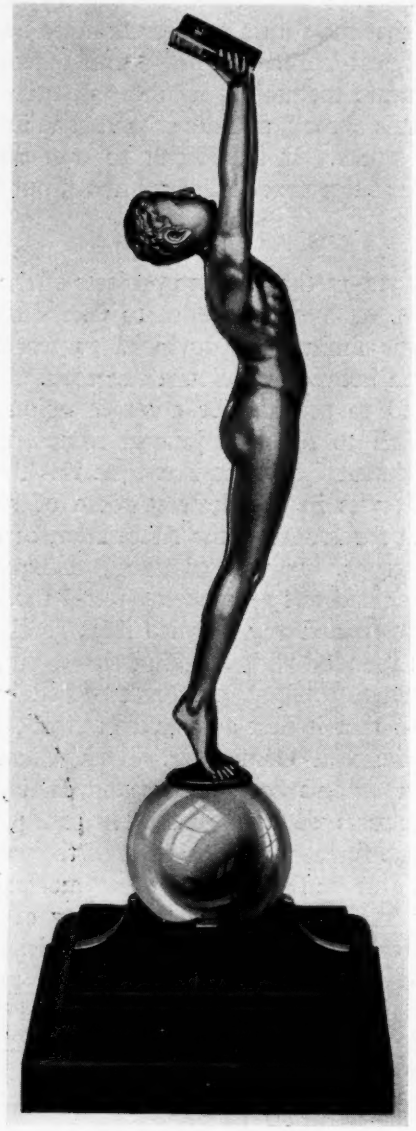
It was in the years 1912–13 that the two projects of the Association of Surgeons and the BRITISH JOURNAL OF SURGERY began to take shape. The membership of the Chirurgical Club was limited to provincial surgeons. Its *raison d'être* was to bring closer association between provincial surgeons. This having been accomplished, it then remained to provide for a closer union between provincial and metropolitan surgeons and to found a journal representative and expressive of British surgery. Sir Rickman Godlee at that time was the President of the Royal College of Surgeons. It was in the drawing-room of his house that Moynihan first mooted his proposal for founding the Association of Surgeons to an audience of leading London surgeons. It was accepted, though with some doubt and criticism. But the matter had not proceeded beyond the stage of preliminary discussion when the war postponed any decisive step, so that it was not until 1919 that the Association was founded with Sir John Bland-Sutton as its first President.

Fortunately the BRITISH JOURNAL OF SURGERY had been planned and started before the war began, so that it first appeared in April, 1913, with Moynihan as the Chairman of the Editorial Committee. That Committee has always been representative of all the teaching schools of England, India, and the Dominions. It was faced with a momentous decision in August, 1914, when the proposal was seriously made to postpone further publication until the war was over. It was then thought that this would only be for a period of at most six months! But it was decided to "carry on" and to seize the opportunity of recording the history of British surgery in the war. This was a fortunate decision, because it brought us into close touch with Belgian, French, and American surgeons. Moynihan was then the Chairman of the Consulting Surgeons attached to the British Army, and his wise guidance of the BRITISH JOURNAL OF SURGERY helped to establish the new venture in a position of assured success.

During the six years of his Presidency of the Royal College of Surgeons, Moynihan lost no opportunity of encouraging the promotion of surgical research, both at the laboratories of the College and later on at the Buckston Browne Farm.

The JOURNAL has been born, it has survived the dangers of infancy and adolescence, and grown to adult age during the more than twenty-one years that Moynihan has presided over its destinies. During that time he never missed a meeting of the Editorial Committee, and these meetings have always been as happy under his chairmanship as they have been fruitful of good results.

It is in grateful recognition of the great part which Lord Moynihan has played in the founding and editing of this JOURNAL and in the promotion of surgical research that a testimonial is presented to him by the Editorial Committee and by the subscribers to the BRITISH JOURNAL OF SURGERY. This takes the form of a



silver statuette of symbolic character designed and made by Mr. Omar Ramsden; of the gift of a thousand guineas to the Council of the Royal College of Surgeons for the promotion of research; and of a tablet placed on the walls of the College perpetuating our sense of indebtedness to a great surgeon and a great leader.

TOTAL REMOVAL OF A LUNG

By R. MILNES WALKER
HONORARY SURGEON, ROYAL HOSPITAL, WOLVERHAMPTON

THE subject of total removal of a lung is one which has had increasing prominence in the literature during the last few years. Though the operation was first attempted as long ago as 1910, no successful case was reported until 1931. Up to the present there have been reports of 34 cases in the literature and references to a further 10. Of 36 cases where the diagnosis is given, in 19 the operation was performed for a tumour of the lung, and in the remaining 17 for inflammatory conditions. Of the total of 44 cases, 24 have survived the operation, giving a case mortality of a little under 50 per cent. These results are steadily improving, and the figures for the last year or two are considerably better than this.

The problems connected with this extensive operation have recently been admirably summarized in a paper by Overholt,[1] who gives a careful description of a series of 8 personal cases. There is no doubt that in the present limited experience of the operation no definite rules can be laid down. The present writer will, therefore, enumerate the main problems connected with this operation, particularly as performed for inflammatory conditions in the lung, and give a report on a personal case together with a table of the cases so far recorded. It was felt that such a table might throw light on some of the more important dangers of the operation, with particular reference to the fatal cases where the cause of death is stated.

There are two separate groups of cases in which this operation of removal of a whole lung has been performed: firstly, bronchiectasis and multiple abscesses in the lung, and, secondly, new growths. In cases of bronchiectasis, it is necessary to ascertain by means of a bronchogram that the disease is entirely limited to one side, but in those cases in which both upper and lower lobes are involved, and which have not yielded to less severe measures, the operation certainly has a field of usefulness; whether it will displace a thoracoplasty for such cases remains to be seen after further experience, but most thoracic surgeons seem to be agreed that the results of the latter operation in this disease are disappointing. Regarding malignant growths of the lung, the increasing use of the bronchoscope is certainly leading to earlier diagnosis, and where there is a possibility of radical extirpation of the disease by means of pneumonectomy, the attempt is certainly worth making. How permanent as regards the question of recurrence the results will be, can only be ascertained after a much longer experience, but the supervention of both local recurrence and cerebral metastases has already been recorded in cases where it had been hoped that the disease had been entirely eradicated by complete removal of the lung.

The problem is somewhat different in the inflammatory cases from what it is where a new growth without subsequent inflammation exists. No definite rule can be laid down regarding the choice of a single-stage or a multiple-stage operation;

in most of the early cases a multiple-stage procedure was employed, but the recent tendency is towards completing the whole operation at a single sitting. The presence of only one fatality from shock in the whole series (in a tuberculous case with very extensive adhesions) leads one to consider that with adequate means for intravenous infusion or blood transfusion, patients stand this extensive operation remarkably well, and the need for performing the operation in multiple stages will be confined to those cases only in which there are very dense adhesions.

In some respects the problems are similar to those confronted in cases of lobectomy, but there is the important difference that in lobectomy the remaining portion of the lung still occupies the pleural cavity, and steps have to be taken to maintain its expansion and to encourage it to fill the whole cavity on that side, whereas in pneumonectomy there is the need for the obliteration of this cavity.

One of the first considerations is the necessity for preliminary pneumothorax; here the problem closely resembles that in lobectomy, but the writer feels that where a pneumothorax is possible it should be carried out ten to twelve days before the major operation. In cases of bronchiectasis, however, such as the one now recorded, the adhesions between the lung and chest wall frequently make this impossible. In the inflammatory cases the freeing of these adhesions may occupy the greater part of the operation; in these cases also the structures of the hilum may be matted together by fibrous tissue, so that their dissection and individual ligature is impossible. Where the operation is undertaken for a new growth, however, there are not likely to be adhesions, unless an inflammatory process has supervened, and the approach to the hilum is not nearly so difficult; it is in these cases that the anterior incision has been found to give good access to the hilum, whereas in the inflammatory cases the postero-lateral route appears to be more satisfactory.

As regards the anæsthetic, the writer has used nitrous oxide and oxygen for both lobectomy and total pneumonectomy. Postural drainage is carried out immediately before the patient is taken to the operating theatre, in order to drain the lung as completely as possible. Where there is little or no sputum there appears to be no need for intratracheal intubation, and in the present case the anæsthetic proved quite satisfactory with a closed mask and positive pressure, without intubation. The lateral movements of the mediastinum, however, were somewhat alarming during the course of the operation, but this was steadied to some extent by the assistant by means of a clamp on the diseased lung during the early part of the operation, and by fixation of the tourniquet on the pedicle, after this was applied. The postero-lateral approach was used and gave a very satisfactory exposure of the hilum, a long incision being made in the 6th intercostal space. The residual space was drained by means of closed drainage and a water seal, which seems to be a necessity where there has been risk of infection of the pleural cavity, though a number of cases are recorded, particularly where the operation has been performed for neoplasms, in which primary closure of the chest has been effected, and aspiration of serum performed only when necessary afterwards.

The necessity for a subsequent thoracoplasty appears to depend on a number of factors, but the present case shows that it is by no means necessary in all instances, and, particularly in children, the chest wall and mediastinum are capable of adapting themselves to the changed conditions without either a phrenicectomy or a thoracoplasty.

The means of dealing with the hilum is a matter which is by no means settled, but is perhaps the most fundamental step of the operation. All grades of technique are being practised by different observers. First of all there is mass ligature of the hilum, allowing the lung to separate naturally, which is apt to happen after about fourteen days. This method, which was practised by Nissen in the first case of successful pneumonectomy, and has since been followed by Mason, has the advantage that it reduces the time of the operation and does away with the danger of primary hæmorrhage from the pulmonary vessels. It entails, however, the necessity of leaving a widely open wound, containing the necrotic lung, with infection of the pleural cavity, and the certainty of a bronchial fistula when the lung separates. Subsequent anæsthetics may be necessary for dressing the wound, and in one case the lung, which had failed to separate after thirty days, had to be removed with the cautery. Then there is the mass ligature of the hilum with primary amputation of the lung; this is also very prone to give rise to a bronchial fistula and infection of the residual space. The next method is the application of a tourniquet and suture of the pedicle with mattress sutures; this has been practised successfully by Roberts and Tudor Edwards, and was performed in the author's case without any subsequent bronchial fistula. A Shenstone-Janes tourniquet of silk was used, and the lung amputated by diathermy $\frac{1}{4}$ in. distal to the tourniquet, the short thickened pedicle making it impossible to apply a second tourniquet on the pulmonary side; any escape of pus from the bronchi was carefully prevented by means of a swab. Five mattress sutures of catgut were then inserted right through the pedicle, and the tourniquet removed. A few further catgut stitches were put in, approximating as far as possible the mediastinal pleura in front of and behind the pedicle, though there was insufficient free pleura to cover the stump completely; the bronchial stump had retracted to some extent but was not entirely buried. Judging from the writer's and others' experience of lobectomies, primary closure of the bronchial stump may be obtained by this method in the majority of cases. The fourth method of dealing with the hilum is the dissection of its individual structures, and their separate ligature. The risk in this is injury to the pulmonary artery, and a number of cases are recorded where fatal hæmorrhage from this artery has occurred while this method was being practised. Its advantage in cases of malignant disease is obvious in that the bronchus may be ligatured more proximally and glands can be dissected out from the hilum, but it is particularly dangerous in cases of inflammatory disease of the lung, where there is apt to be considerable fibrosis among the structures of the hilum, and in these cases it appears to have no advantages over the tourniquet and suture method.

CASE REPORT

Male, aged 7 years, was first admitted to the Royal Hospital, Wolverhampton, in June, 1929, at the age of 11 months. There was a history of cough for three months, and difficulty with breathing for six weeks. A diagnosis of bronchitis was made, and after a fortnight he returned home. Four months later he was re-admitted with signs suggesting bronchopneumonia in the left lower lobe; this subsided, but the cough persisted, and he continued to attend the hospital, being an in-patient again in November, 1930, when he is reported to have had consolidation of the left lower lobe; on being re-admitted for the fourth time four months later, the consolidation was again noted at the base of the left lung, but there was no fever. In December, 1930, both antra, which contained mucopus, were washed out, and the following month the tonsils and adenoids were removed. He was not in hospital again for

four years, but during that period the cough continued unabated, and he attended the Out-patient Department spasmodically.

In February, 1935, he came into hospital, having had measles three weeks previously, followed by pains in the chest; the cough was as bad as ever; he was bringing up from 1 to 4 oz. of offensive sputum daily, clubbing of the fingers was present, and there was dullness and impaired entry over the whole of the left side of the chest. On aspiration of the left base, 5 c.c. of purulent fluid were withdrawn, and this, on culture, yielded pneumococci. An X-ray examination showed the left chest to be completely opaque with poor movement; the right lung field was clear, and the heart was displaced a little to the left. During the following two months his condition remained stationary; he had no fever most of the time, but every two or three weeks he had a febrile attack lasting two or three days, the temperature rising to about 101°. In April he had one of these attacks which was much more severe than his previous ones; the temperature rose to 103°, the pulse to 150, and respirations to 56; some crepitations were present at the right base, but the physical signs on the left side remained

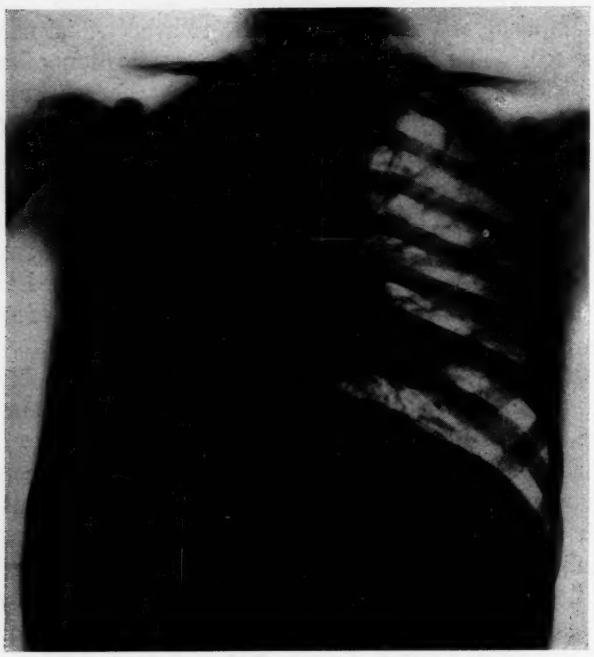


FIG. 1.—X-ray of chest before operation.

unchanged. A further small quantity of pus was aspirated from the left side. On this occasion the fever took two weeks to subside; he then developed an alveolar abscess for which an upper molar tooth was extracted.

A lipiodol injection in May showed bronchiectatic cavities in both upper and lower lobes on the left side, but the right lung was clear. Postural drainage did not improve his condition, and it was decided to attempt a pneumonectomy on the first favourable occasion after he had remained free from a febrile attack for some weeks. Attempts at a preliminary artificial pneumothorax were unsuccessful. *Figs.* 1–3 show the radiographic appearances before and after operation.

OPERATION.—The operation was performed at the Royal Hospital, Wolverhampton, on Sept. 3, 1935. The anæsthetic, administered by Dr. J. H. Richmond, was nitrous oxide and oxygen, without intubation of the trachea, positive pressure being maintained with a closed mask. A long incision was made along the 6th intercostal space, and a small portion of the 6th rib was removed near its angle. The lung was found to be completely adherent, but the adhesions could be separated with the fingers without much difficulty, except at the apex and

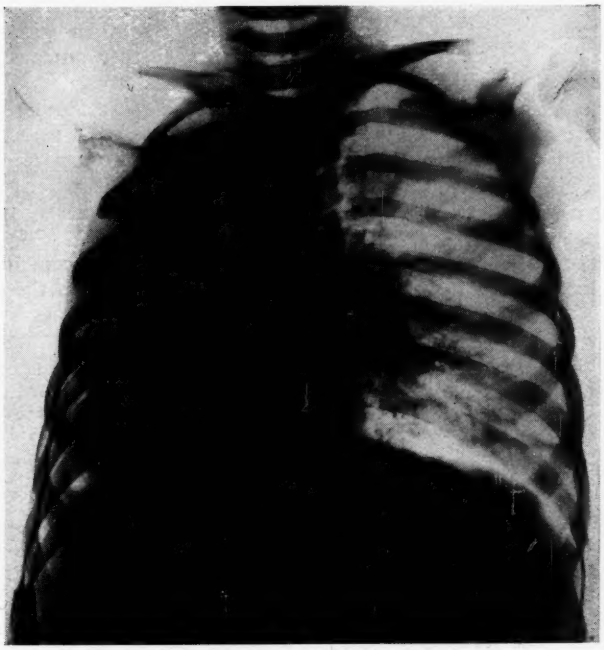


FIG. 2.—Bronchogram before operation.

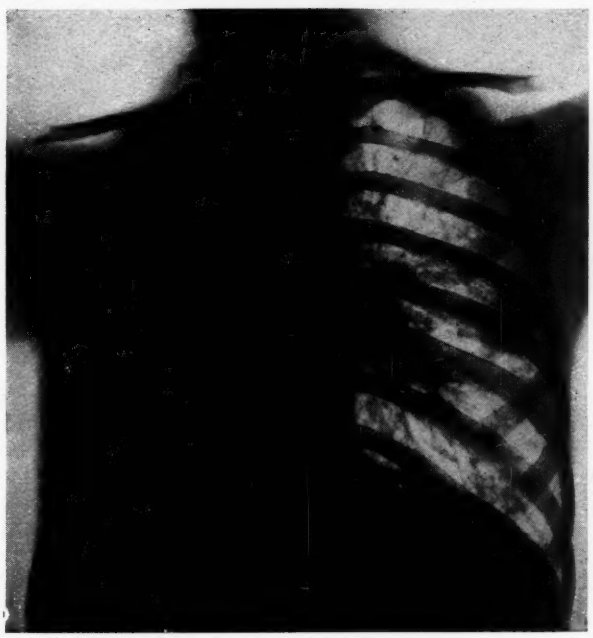
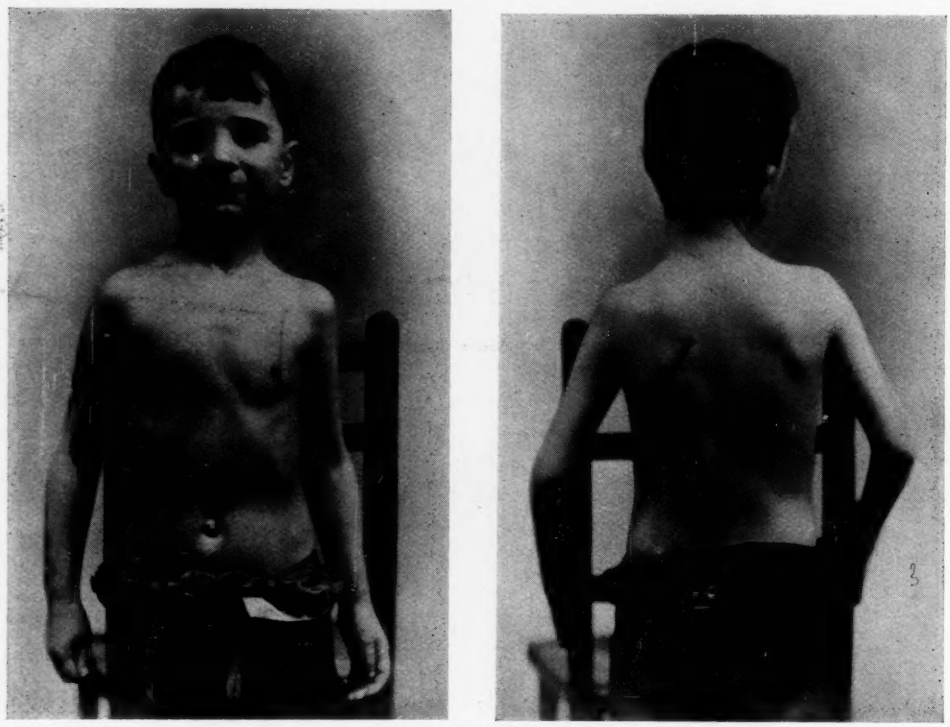


FIG. 3.—X-ray of chest one month after operation.

the diaphragm, where they were divided by diathermy. The lung was then freed on all sides down to the hilum, as close to the mediastinum as possible, and the lung amputated by diathermy ¼ in. distal to the tourniquet. The pedicle was then secured with mattress sutures of catgut, and the tourniquet removed; a few further stitches were then placed to approximate the pleural margins as far as possible, but the stump could not be completely buried. Intercostal drainage was provided by means of a self-retaining catheter through a stab in the 9th intercostal space, and the wound closed with catgut sutures, leaving a small rubber drain into the subcutaneous tissues. The catheter was connected to a water seal. The pulse-rate, which had been 120 at the beginning of the operation, had risen to 140, but there was remarkably little shock, and at no time was it necessary to administer fluids except by mouth.

SUBSEQUENT PROGRESS.—On the fourth day the intercostal drain came out, and no further drainage occurred from the stab-wound; there was a little pus from the main incision, but



FIGS. 4, 5.—The patient three months after operation. The vertical line drawn just outside the left nipple line marks the right border of cardiac dullness.

this appeared to originate in the subcutaneous tissues. Five days after operation the right border of cardiac dullness was ½ in. inside the left nipple line; on the tenth day he coughed up 4 oz. of thick ropy pus, evidently an accumulation round the stump which had discharged itself into the bronchus; apart from this, the sudden cessation of his sputum following the operation was most marked. Two weeks after operation his wound was healed, and he left hospital on Oct. 10, thirty-seven days after operation. He continued to make further improvement, and eleven weeks after his operation was able to make the journey of 250 miles to London and back in the day, to be presented at a meeting of the Royal Society of Medicine.[2] (*Figs.* 4, 5).

At the present time, six months after the operation, he is in good health; he can run about and play with other children without getting breathless, though he has an occasional dry cough, but never brings up any sputum; the mediastinum is displaced to the left, the

right border of cardiac dullness being outside the left nipple line ; the heart appears to have become rotated backwards as well as to the left, for the heart-sounds are very loud over the back of the left chest, and cardiac pulsation can be felt in the 6th and 7th intercostal spaces in the scapular line ; there is slight dropping of the shoulder and flattening of the ribs on that side, but no scoliosis. His weight since the operation has increased from 39 to 45 lb.

PATHOLOGY.—The specimen of the excised lung shows that it is covered with thickened pleura, to which are attached a number of filmy adhesions ; a little adherent fat has come away with the lung at the apex, and the interlobar fissure is quite obliterated ; the inner aspect shows that the hilum has been divided at the point where the left bronchus divides into its two main branches ; the cut section shows the whole lung to be consolidated, with much fibrosis, especially in the lower lobe, and all the bronchi are moderately dilated. (*Fig.* 6.)

FIG. 6.—Vertical section of the excised lung.

A study of *Tables I* and *II* summarizing the cases of total pneumonectomy brings out a number of interesting points. There are references to 44 cases, 17 for inflammatory conditions, 19 for new growths, and 8 in which the diagnosis is not given (Churchill,[22] 8 cases with 4 deaths). Of the total of 44, there have been 20 deaths, a mortality of 45 per cent, but in the 17 inflammatory cases the mortality is 29 per cent, and 59 per cent in the 19 cases of growths. The high mortality in the latter group appears to have a close connection with the more advanced age of the patients, for in those cases where the age is stated, there were 3 deaths among 16 cases under thirty (18·7 per cent), and 6 deaths among 12 cases over this age (50 per cent). Of 14 cases where the cause of death is stated, this was primary or reactionary hæmorrhage (3), secondary hæmorrhage (1), shock (1), mediastinal infection (1), pneumonia or œdema of the remaining lung (3), septicæmia (1), and other causes (4). It will be noted that there has been no death among the last 10 cases of bronchiectasis or pulmonary abscesses recorded. (*See Table III.*)

Table I.—TOTAL PNEUMONECTOMY FOR INFLAMMATORY CONDITIONS

AUTHORITY	DIAGNOSIS	AGE AND SEX	DATE OF OPERATION	ANÆSTHETIC	TREATMENT OF HILUM	RESULT
Meyer[3]	Bronchiectasis, L. ..	44 F.	22/12/15	Local and anæsthol	Structures ligated separately	Died; hæmorrhage from pulmonary artery
Lilienthal[4] ..	Bronchiectasis, R. ..	12 F.	28/ 2/19	Nitrous oxide and ether	Suture	Died; hæmorrhage from pedicle 6 hours later
Lilienthal[4] ..	Bronchiectasis, L. ..	8 F.	17/ 6/20	Nitrous oxide and ether	Suture	Died; secondary hæmorrhage from pedicle 13th day
Nissen[5]	Bronchiectasis, L. ..	12 F.	1931	Ether ..	Mass ligature, lung allowed to slough	Recovery
Windsberg[6] ..	Bronchiectasis, R. ..	12 F.	8/11/32	Avertin and nitrous oxide	Structures ligated separately	Recovery
Archibald[7] ..	Tuberculosis	—	—	—	Mass ligature ..	Died; shock, 2 hours later
Haight[8]	Bronchiectasis, L. ..	13 F.	14/11/32	Nitrous oxide	Mass ligature, lung allowed to slough	Recovery
Alexander[9] ..	Multiple abscesses ..	—	19/ 6/33	—	—	Died; 8th day
Alexander[9] ..	Bronchiectasis, L. ..	—	16/10/33	—	—	Recovery
Mason[10] ..	Bronchiectasis, L. ..	13 F.	4/ 5/34	Avertin and nitrous oxide	Mass ligature, lung allowed to slough	Recovery
Overholt[1] ..	Pulmonary abscess ..	44 F.	8/10/34	Cyclopropane	Suture	Recovery. (Died, pneumonia, 7 months later)
Mason[10] ..	Bronchiectasis, R. ..	18 M.	18/10/34	Avertin and nitrous oxide	Mass ligature, lung allowed to slough	Recovery
Gower and Tudor Edwards[11]	Bronchiectasis, L. ..	7 M.	6/ 2/35	Avertin and nitrous oxide	Suture	Recovery
Roberts[12] ..	Bronchiectasis, L. ..	5 M.	8/ 2/35	—	Suture	Recovery
Overholt[1] ..	Multiple abscesses, L. ..	28 F.	23/ 2/35	Evipan and cyclopropane	Suture	Recovery

Roberts[12] ..	Bronchiectasis, L. ..	5 M.	8/ 2/35	—	Suture ..	Recovery
Overholt[1] ..	Multiple abscesses, L. ..	28 F.	23/ 2/35	Evipan and cyclopropane	Suture	Recovery
Gower and Tudor Edwards[11]	Bronchiectasis, R. ..	10 M.	22/ 7/35	Nitrous oxide	Suture	Recovery
Walker[2]	Bronchiectasis, L. ..	7 M.	3/ 9/35	Nitrous oxide	Suture	Recovery

Table II.—TOTAL PNEUMONECTOMY FOR NEW GROWTHS

AUTHORITY	DIAGNOSIS	AGE AND SEX	DATE OF OPERATION	ANÆSTHETIC	TREATMENT OF HILUM	RESULT
Kummell[13] ..	Carcinoma, R. lung ..	48 M.	10/11/10	—	Clamped; clamp left to separate	Died; 6th day; infection around stump
Meyer[3]	Lymphosarcoma, R. lung ..	5½ M.	16/ 4/20	—	Clamped and sutured	Died; 10 hours later; pulmonary œdema
Archibald[7] ..	Carcinoma, L. main bronchus	34 M.	10/ 2/32	—	Structures ligated separately	Died; 4th day; streptococcal septicæmia
Ivanissevich and Ferrari[14]	Epithelioma, bronchus ..	—	11/10/32	—	—	Died; 8th day
Lilienthal[15] ..	Sarcoma, L. lung ..	34 F.	28/ 1/33	—	Structures ligated separately	Died; 4th day; bronchopneumonia
Graham and Singer[16]	Carcinoma, L. upper bronchus	48 M.	5/ 4/33	Nitrous oxide	Structures ligated separately	Recovery
Archibald[7] ..	Sarcoma, L. upper lobe ..	31 M.	7/ 7/33	Spinal and nitrous oxide	Structures ligated separately	Recovery. (Died of recurrence 6 months later)
Reinhoff[17] ..	Benign tumour, L. bronchus	3½	24/7/33	Avertin and nitrous oxide	Structures ligated separately	Recovery
Overholt[18] ..	Carcinoma, R. lung ..	33 F.	2/11/33	Cyclopropane	Structures ligated separately	Recovery. Well 18 months later

Continued on next page

Table II.—TOTAL PNEUMONECTOMY FOR NEW GROWTHS, *continued*

AUTHORITY	DIAGNOSIS	AGE AND SEX	DATE OF OPERATION	ANÆSTHETIC	TREATMENT OF HILUM	RESULT
Reinhoff[17] ..	Carcinoma, L. primary bronchus	42 F.	3/11/33	Avertin and nitrous oxide	Structures ligated separately	Recovery
Freedlander[19] ..	Carcinoma, L. main bronchus	—	4/11/33	—	—	Died following subsequent thoracoplasty
Alexander[9] ..	Carcinoma, L. bronchus ..	—	6/11/33	—	—	Died; cardiac failure, 30th day
Overholt[1] ..	Carcinoma, L. lower bronchus	33 F.	2/ 5/34	Ethylene ..	Suture	Recovery. Well 1 year later
Overholt[1] ..	Carcinoma, L. lung ..	43 M.	7/ 7/34	Cyclopropane	Suture	Died; 48 hours later; hæmorrhage from pulmonary artery invaded by growth
Overholt[1] ..	Carcinoma, L. main bronchus	37 M.	13/11/34	Cyclopropane and ethylene	Suture	Died; hæmorrhage from retracted pulmonary artery during operation
Overholt[1] ..	Carcinoma, R. lung ..	50 M.	8/12/34	Cyclopropane	Suture	Died; 6th day; pneumonia
Overholt[1] ..	Carcinoma, L. upper bronchus	59 F.	8/ 4/35	Cyclopropane	Structures ligated separately	Recovery
Graham[20] ..	Carcinoma	—	—	—	—	Died; 3½ weeks later, aspiration of fluid from bronchial fistula
Haight[21] ..	Carcinoma	—	—	—	—	Recovery

Table III.—Mortality in Relation to Age
(28 Cases)

Age	Recovered	Died
Years		
0– 9	5	2
10–19	6	1
20–29	2	0
30–39	3	3
40–49	2	2
50–59	1	1

It is yet far too early to assess the full value of this operation, and no prognosis can be given regarding the expectation of life with only one lung. In the case here recorded, however, the child's parents already feel that the operation has been well worth while, for both they and the patient have good nights' sleep instead of being disturbed, as they have been for five years, by his incessant coughing.

REFERENCES

[1] Overholt, R. H., *Jour. of Thoracic Surg.*, 1935, v, 54.
[2] Walker, R. M., *Proc. Roy. Soc. Med.*, 1936, xxix, 212.
[3] Meyer, W., *Arch. of Surg.*, 1923, vi, 361.
[4] Lilienthal, H., *Ann. of Surg.*, 1922, lxxv, 257.
[5] Nissen, R., *Zentralb. f. Chir.*, 1931, lviii, 3003.
[6] Windsberg, E., *Jour. of Thoracic Surg.*, 1934, iv, 231.
[7] Archibald, E., *Ann. of Surg.*, 1934, c, 796.
[8] Haight C., *Surg. Gynecol. and Obst.*, 1934, lviii, 768.
[9] Alexander, J. Quoted by Haight[8].
[10] Mason G. A., *Brit. Med. Jour.*, 1935, i, 299.
[11] Gower, F. J. S., and Edwards, A. Tudor, *Proc. Roy. Soc. Med.*, 1936, xxix, 221.
[12] Roberts, J. E. H., *Ibid.*, 220.
[13] Kummell, H., *Verhandl. d. deut. Gesellsch. f. Chir.*, 1911, xl, 147.
[14] Ivanissevich, O., and Ferrari, R.C., *Bol. y. Trab. de la Soc. de Cir. de Buenos Aires*, 1933, xvii. Quoted by Haight.[8]
[15] Lilienthal, H., *Jour. of Thoracic Surg.*, 1933, ii, 600.
[16] Graham, E. A., and Singer, J. J., *Jour. Amer. Med. Assoc.*, 1933, ci, 1371.
[17] Reinhoff, W. F., *Bull. Johns Hopkins Hosp.*, 1933, liii, 390.
[18] Overholt, R. H., *Jour. of Thoracic Surg.*, 1934, iv, 196.
[19] Freedlander, S. O. Quoted by Haight.[8]
[20] Graham, E. A., *Jour. of Thoracic Surg.*, 1935, v, 78. Discussion on Overholt's paper.
[21] Haight, C., *Ibid.*, v, 79.
[22] Churchill, E. D., *Ibid.*, 80.

HYDATID DISEASE OF THE KIDNEY

By R. CAMPBELL BEGG, Wellington, New Zealand

Even in countries where hydatid disease is common the kidney is affected in only a small percentage of patients. It is proposed in this article to report four cases together with a description of the renal echinoccosis in general and a review of the literature, more particularly that of the last ten years. For a systematic exposition of the subject of hydatids the reader is referred to the works of authorities, of whom Dévé, of Rouen, and Dew, of Sydney, occupy a foremost place. A brief sketch of the nature of the parasite and its mode of access to the kidney is, however, desirable before dealing more fully with the pathological manifestations in the urinary tract.

THE ECHINOCOCCUS IN GENERAL AND ITS MODE OF ACCESS TO THE KIDNEY

*Tænia echinococcus** or *Echinococcus granulosus* is one of the smallest of the Cestodes. Midget among its fellows of the tapeworm tribe, it gains secure refuge from the stress of the current and peristaltic motion of the canine intestine by lodging between the villi like a ship in dock. Minute as a fly's leg, only the base of its terminal proglottid presents to the bowel lumen. Hundreds of white spots amidst the dull red of the mucosa indicate its presence. Ceaselessly it devotes itself to the task of reproduction. Self-impregnated, the proglottid swells as its ovary fills to bursting point. Like a loaded vessel it casts off into the stream, the vacated berth being taken by the next of the series. Thousands of these ripe proglottides, each with its hundreds of ova, pass to the outer world to infest the pastures and pass into the stomachs of the grazing herbivora of the neighbourhood. Other eggs become attached to the hair and muzzle of the dog, ready to be transferred to the fondling hand and thence to the intestine of the farmer's child. Inspiration of the eggs as a method of infection was suggested by Dougan Bird in 1877, but is now considered exceptional, though Dévé has produced the disease experimentally by this route. In the slaughter-houses of Split, in Dalmatia, 85 to 100 per cent of the infected sheep—and nearly all are infected—have cysts only in the lungs (Račic). We are really as yet uncertain on the question of respiratory infection and, indeed, ignorant of the method by which the onchosphere attaches itself to the wall of the bowel and penetrates it to gain the circulatory system and the tissues. Dew observed the *fait accompli*, embryos in the portal veins of swine a few hours after

* In addition to *Echinococcus granulosus*, the common form, other species have been described—*E. oligarthus* in the jaguar (Diesing), *E. minimus* in the Macedonian wolf (Cameron), and *E. longimanubrius* in the Cape hunting-dog (*Lycaon pictus*). No cystic forms of the last three have been identified except perhaps of *E. oligarthus* in the agouti or American guinea-pig. (T. W. M. Cameron, "Some Modern Biological Conceptions of Hydatid", *Proc. Roy. Soc. Med.*, 1926–7, xx, Pts. 1 and 2, 273).

ingestion. Having reached the blood-stream, the little animal is small enough to pass through the capillary system of the liver should it choose one of the wider capillary anastomoses. Its projecting hooklets may hold it up like an oxalate calculus in a ureter, or it may have a selective preference for the liver, for it stays there in the vast proportion of cases. A fair number, however, win through the right heart to the lung, though K. D. Fairley, impressed with the frequency of lower right lobe pulmonary cysts in man, revived the direct migration theory of Küster. Should the hexacanth pass through the lung filter, it reaches the left heart and has then all the organs and tissues of the body open to its invasion. The kidney is a favoured site, but cysts in it form a meagre 0·5 to 4 per cent of the whole in the various published series of cases.

Other routes to the kidney have been contemplated—by the thoracic duct and left subclavian vein, missing the liver; by the venous plexus of Retzius (Nicaise); and by direct migration from the duodenum or colon (Küster). The not infrequent occurrence of one cyst only in the body, and that in the kidney, instead of a series of liver and lung cysts strewn along the route of migration, gives ground for consideration of other than the usually accepted route; or, perhaps, there is a selective preference for one organ or the other—certain groups finding a liver or lung environment uncongenial. In the ultimate site of lodgement the embryo sheds its hooklets, and proliferates, secreting its fluid internally and its hyaline laminated membrane externally, and becomes the familiar hydatid cyst.

Eggs must often be swallowed in clumps, and at more than one time by those exposed—a charge of small shot, not a bullet. It follows that many hexacanths must perish—swept out of the bowel or destroyed in the tissues—for one that survives. One cyst developed may act as a preventive for future infection in virtue of produced antibodies.

The embryo having reached the kidney, the amazing process of the second stage of reproduction begins. In man it is the last. The final stage of evolution depends on the consumption by the definitive host of the intermediate one. The cycle is between carnivora and herbivora. In the former the worm is in the intestine to be discharged, in the latter in the tissues to be eaten. Sheep and dogs provide the necessary field. Man is a side line. More fastidious than its big brother *Tænia solium*, which may be found in both phases in man, the adult echinococcus is never a parasite of the latter. Scolices swallowed or ruptured into his intestine pass out without further development.

The reproductive urge, however, goes on unabated. The whole system of quivering opalescent mother cyst, brood capsules, sand, pearl, or ruby daughter cysts, third generations, and separated scolices develops in the human subject, as in the animal. The potency of propagation staggers the imagination. Thousands of worms in one dog each producing a series of ripe proglottides in endless continuity; each proglottid with its hundreds of eggs; each egg able to produce an embryo and form a cyst; every cyst with its multitude of brood capsules and scolices (Dévé found 400,000 of the latter in a single cubic centimetre of hydatid sand); each scolex able to produce yet another cyst in the same host, with a geometrical progression of numbers, *ad infinitum*.

The scolex has indeed two lines of development open to it according to circumstances: in the tissues of the intermediate host, another cyst; in the intestine of the dog, a full-sized worm.

FREQUENCY AND DISTRIBUTION OF RENAL HYDATID CYSTS

Nicaise, writing in 1914, had collected 474 cases of renal hydatid—377 from the literature and 97 received on invitation from doctors in many countries. Between that date and 1925, collective studies were published by Kretschmer, Russell and Kilbane, and others. Inclusive of a few pararenal cases noteworthy for their intimate relation to the kidney, the reports of the last ten years (1925–35) are given in the following table. It was compiled as the result of a fairly thorough search and may be considered as reasonably exhaustive :—

Country	No. of Articles Published	No. of Cases
Europe—		
Great Britain	4	4
France	6	9
Germany	4	3
Spain	3	3
Italy	12	24
Austria	1	3
Hungary	3	3
Holland	1	1
Czechoslovakia	1	1
Bulgaria	1	1
Rumania	2	2
Jugoslavia	2	10
Greece	1	46
Ukraine (U.S.S.R.)	5	5
U.S.S.R. (excluding Ukraine)	4	69
	— 50	— 184
Asia—		
Iraq	1	2
Siberia (U.S.S.R.)	2	2
Trans-Caucasia (U.S.S.R.)	1	1
Central Asia (U.S.S.R.)	1	6
	— 5	— 11
North America—		
United States	11	13
	— 11	— 13
South America—		
Argentine	13	17
Uruguay	3	3
Chile	1	1
	— 17	— 21
Africa—		
French North Africa (Tunisia and Algeria)	2	37
	— 2	— 37
Australia	4	20
	— 4	— 20
New Zealand	1	1
	— 1	— 1
Total	90	287

In this table the widespread distribution is indicated, but not the actual incidence. Iceland, once the most infested country, is not represented, though a great deal has been added to its hydatid history since the classic work of Finsen (1867). In richly journalized North America few of the scanty store of cases have escaped publication. Infested Arabia produced only two cases. China, Japan, and most of the smaller republics of America provide no reports. Taken as a whole, most cases no doubt remain unpublished. The interested student is especially commended to the collected series in the list: Gall (Russia, 66 cases); Toole (Greece, 46 renal and 10 pararenal); Goinard (French North Africa, 36 cases); and to the authors with most personal cases: Craig and Lee-Brown (16), Vila (8), Videnski (6), Oulié (4), Blum (4), and Račić (10).

A few articles in this decennium appeared without cases. They will be indicated in the appropriate place.

The world's literature contains a few reports of hydatid disease of the kidney every year. The list of 287, after subtracting collected cases, some of which were doubtless previously published, gives at least 161 new cases, making with the Nicaise series, and the twelve years subsequent, a total of approximately 800 cases, so that the features of this rare complaint are completely available for study.

The incidence of the disease fluctuates in a given country with the number of sheep, the living conditions, level of culture, and the efficacy of preventive measures. It is decreasing in Iceland and France, increasing in Siberia, the Argentine (Greenway), Greece (Toole), and Dalmatia (Račić). In England, though rare in man, it is said to be prevalent among sheep (Brailsford). In New Zealand it is keeping pace with the increase of population (Barnett).

NOMENCLATURE

The general build of an hydatid cyst is the same in all soft organs. The nomenclature has been somewhat confused, but the terms as here defined will be used throughout the article.

Pericyst.—This is the adventitia or boundary wall formed by the fibrocellular reaction of the tissues of the host.

Cyst.—The cyst is the cavity bounded by the pericyst with its contents.

Ectocyst.—The ectocyst is the limiting envelope of the parasite proper. The term will be used to embrace both the hyaline laminated membrane and the germinal layer within it. Considered separately the latter is sometimes known as the endocyst.

Pericystic Cavity.—This is a coined term to denote the potential and sometimes actual space existing between the pericyst and the ectocyst.

Endocystic Cavity.—This expression will be used to indicate the space bounded by the ectocyst. Its contents are hydatid fluid with brood capsules, free scolices and hooklets, and sometimes daughter cysts and their contents.

RELATION OF CYSTS TO THE RENAL PELVIS

There are three classes of renal cyst according to the relation maintained to the kidney pelvis—namely, open, closed, and pseudo-closed. In the first both pericystic and endocystic cavity communicate with the pelvis, in the second neither, and in the third only the pericystic cavity, the ectocyst remaining unruptured.

Characteristic symptoms are found corresponding roughly to this classification. In the open cysts—renal colic and the passage of hydatid elements in the urine; in the closed the symptoms are mostly due to pressure; in the third the typical manifestations are intermittent attacks of localized loin pain due to calix colic, and perhaps urinary symptoms such as hæmaturia or frequency, but no hydatid material is passed.

CASE REPORTS

Of the four cases only the first lent itself to a systematic study. In the second a destroyed kidney was removed twenty-three years after the original operation for hydatid disease. In the third the patient refused investigation. In the fourth the diagnosis was confirmed at autopsy. The second and third cases are only given in brief.

Case 1.—Female, aged 38. Single.

HISTORY.—The patient had lived in sheep country all her life. She suffered from left renal colic at the age of 18. Passed some membranous material unidentified. No further trouble. At the age of 21 and again at 24, laparotomy was performed for vague abdominal pains. A small corpus luteum cyst was removed at the first operation, a normal appendix at the second. Thereafter the patient was perfectly well till seven days before consultation. One evening she experienced agonizing radiating pain in the left loin. This passed off and next morning she had retention of urine. After being relieved by catheter, she passed a "white grape-like body". Her medical attendant found this to be hydatid membrane and referred her with the diagnosis of hydatid cyst of the left kidney.

FIG. 7.—*Case* 1. Pyelographic appearances.

ON EXAMINATION (July 12, 1935).—The patient's general condition was good and all organs were normal. Neither kidney painful, tender, or palpable.

Urine.—Specific gravity 1020, acid. No abnormal elements, chemical or bacteriological.

Plain X-ray.—Normal-looking kidney shadows. No opacities from calcification.

Cystoscopy.—Ureteric orifices normal on both sides. Some bulging inwards of the posterior bladder wall.

Pyelogram (abrodil—half strength 6 c.c.).—Left pelvis and ureter normal in size and position. The upper major calix is interrupted in its course between pelvis and minor calices by a broad expansion in which blotches and patches of shadow appear here and there. At the top of this area are two well-defined and normal minor calices. The middle calix ends abruptly and a faint ribbon of shadow passes upwards from it behind the blotchy area of the upper calix. The lower calix divides into two branches. The upper of these is normal with a minor calix showing at its extremity. The lower branch is somewhat club-shaped and wider than usual. Two normal minor calices appear at its outer termination. The shadow in this lower major calix is uniform, but perhaps a little fainter than that in the main part of the pelvis. (*Fig.* 7.)

The intravenous pyelograms demonstrated good and equal function of both kidneys, but no pelvic abnormality was apparent.

DIAGNOSIS.—Hydatid cyst of upper pole of the left kidney. The blotchy area was considered to be the cavity of the cyst communicating with the upper calix. No explanation of the normal minor calices opening into the cyst could be suggested until the specimen was examined after nephrectomy.

OPERATION.—Left nephrectomy through a loin incision extended well forwards. No cyst was apparent until the upper pole was reached, when a whitish area was seen at the upper and lateral angle. Dense adhesions to suprarenal capsule and diaphragm. A small portion of the suprarenal capsule was removed with the kidney. The wound was closed except for a wick of rubber dam.

POST-OPERATIVE COURSE.—Primary healing, followed by discharge of a little pus tracking from where the adhesions were separated. Discharged on the twentieth day, with final healing a few days later. In good health after three months.

Comment.—The case up to this point displayed the usual features of an open hydatid cyst of the kidney. The chief interest lay in the examination of the specimen. This showed :—

1. That daughter cysts were present in the middle calix, the pelvis proper, and the ureter, although for three weeks before the operation there had been no colic, pain, or tenderness in the kidney or ureter.

2. That there were three definite mother cysts in the kidney: (*a*) A primary cyst in the cortex of the posterior part of the upper pole, recently ruptured into the middle calix; (*b*) A secondary but well-established cyst, with intact mother ectocyst, and containing daughter cysts, in the upper major calix; (*c*) A younger cyst with intact ectocyst and containing daughter cysts in the lower major calix.

3. That the urine from the upper and lower poles was conducted to the pelvis by normal papillæ which entered the upper and lower major calix discharging between the ectocyst and the pericyst, the latter in this case being the thickened wall of the calix.

4. That the lower cyst was so deeply situated within the kidney that its presence was discovered only after complete section of the organ.

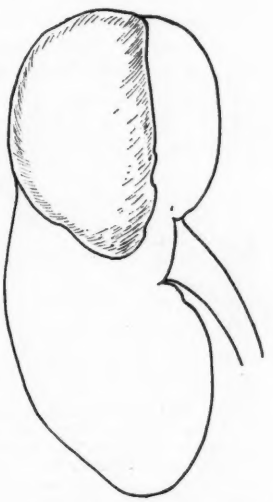


FIG. 8.—*Case* 1. Diagram showing the extent of involvement of the kidney by the cyst (posterior surface).

The importance of such findings in a woman who had been relatively free from symptoms for twenty years, and who had no doubt carried the parasite for thirty, warrants a more detailed description of the findings.

DETAILED DESCRIPTION OF THE SPECIMEN.—

External View (*Fig.* 8).—The kidney was not enlarged and appeared normal, except for the portion overlying the cyst. The latter projected very little from the surface. From the front it was seen as a whitish area with tags of separated adhesions attached, occupying the upper and lateral corner. From behind, a rather larger area was involved reaching almost to the middle of the organ in a downward direction, but affecting only the lateral half. The medial half of the kidney including the upper margin was not involved.

The specimen was bisected in the usual way (*Fig.* 9). The posterior half showed two cysts, one in the upper pole, and one in the lower, quite unconnected with each other. The mother ectocysts of both were intact. Each contained many daughter cysts of the nearly uniform size of large peas. Free in the pelvis lay two cysts, one at the pelvi-ureteral junction. They were both collapsed. At the lower part of the stump of the ureter just above the ligature and 3 in. from the pelvi-ureteral junction lay a third collapsed cyst adhering loosely to the wall of the ureter. A fourth cyst could be seen peeping from the middle calix, which had not been fully exposed with the section.

Mother ectocyst and daughter cysts were removed and the condition shown in *Fig.* 10 revealed.

The upper cavity had rugged fibrous walls, which varied from 1 to 2 mm. in thickness and showed yellow patches of calcification. Above, it reached the kidney surface. In front, two normal papillæ serving the kidney cortex of the anterior part of the upper pole projected

FIG. 9.—*Case* 1. Kidney sectioned showing the two calix cysts with their contained daughter cysts. Both ectocysts are intact. The small cyst seen in the pelvis has come from the middle calix, the upper corner of which may be seen just below the cyst of the upper calix. Papillæ may be seen in relation to both cysts.

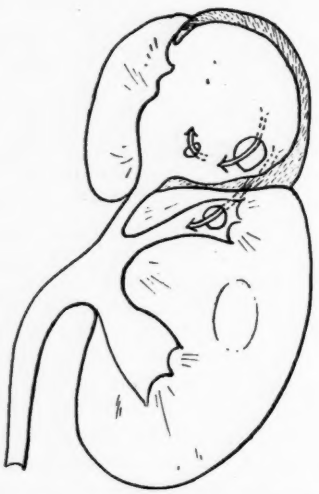


FIG. 10.—*Case* 1. Diagram showing the two orifices of communication between the cortical cyst and that of the upper calix. The lower arrow shows the route by which the contents of the cortical cyst reached the middle calix.

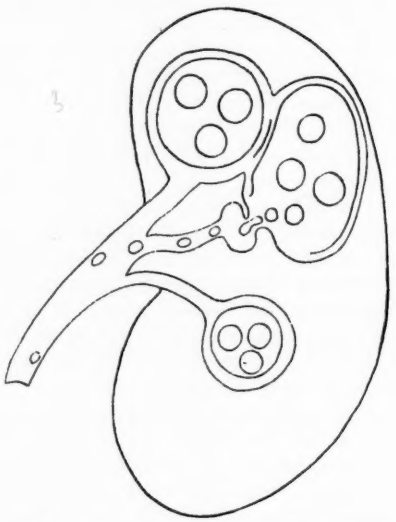


FIG. 11.—*Case* 1. Schematic drawing of the arrangements found in the specimen. The upper and lower calix cysts are closed. Daughter cysts are seen escaping from the middle calix.

into it. They had abutted on the ectocyst, and the urine from them had passed down between it and the adventitia to reach the pelvis proper. The cyst was clearly the upper major calix. Posteriorly, this cavity, which served the dual rôle of calix and cyst, communicated with another cystic cavity through two orifices, well-defined oval rings, occupying the position where normally the papillæ serving the posterior part of the upper pole emerge into the upper major calix. These orifices had all the appearance of being the fibrous rings of the fornices from which the papillæ had been absorbed.

The cavity into which they led was an excavation of the cortex. It was filled with loose daughter cysts of various sizes from clear tense pearls to collapsed pea-sized cysts. A crumpled mass of membrane, probably the shrunken mother ectocyst, lay in a recess (*Fig.* 11). This was the primary cortex cyst. Its contents were prevented from passing into the calix cyst by the tension of the cyst contained in the latter, which occluded the orifices of communication. The cyst wall reached the posterior surface and projected slightly from it. It was thick and fibrous, and in parts calcified, similar to the walls of the calix cyst. Below, it had undermined the base of the papillæ which belonged to the middle calix. One of these papillæ was a mere shell containing a cyst with clear fluid (not hydatid), another had been completely absorbed, and through the fibrous ring of its fornix the cortical cyst communicated freely with the middle calix. The latter was stuffed in every corner with small intact and larger collapsed cysts one of which was projecting into the main pelvis. It was clear that the rupture into the middle calix had provided the material which the patient had passed. These two upper cysts with their thick rugged and partially calcified walls were of undoubtedly long standing.

Lower Calix Cyst.—This was much younger than the other two cysts, though the presence of daughter cysts indicated that it was not a thing of yesterday. The epithelial wall of the calix was little thickened, though white and sclerosed. Normal papillæ from the lower pole projected into the lumen, the conditions being precisely the same as in the upper calix. Two of the cysts, those in the calices, illustrated the condition, which for lack of a better term, I have called 'pseudo-closed'. The pericystic cavity communicated with the pelvis, the endocystic cavity was closed off. The cortical cyst had belonged to the same class, the pericystic cavity communicating both with the upper and possibly with the middle calices. It had, however, recently become an open cyst by rupture of the pericyst or ectocyst into the middle calix.

The puzzling feature of the pyelogram received its explanation. The indefinite blotchy area was not abrodil in the cyst, but in the narrow pericystic cavity between ectocyst and calix wall. By this route it reached and defined the two upper minor calices.

The uniform filling of the lower pole cyst indicated a wider and looser space between ectocyst and calix wall obscuring the appearance of filling defect. The ribbon of shadow from the middle calix led to the ruptured cortical cyst.

PATHOGENESIS.—The series of events may be reconstructed as follows:—

Lodgement of the hexacanth in the cortex of the posterior part of the upper pole.

Absorption of secreting tissue as far as the capsule behind, and the base of the upper papillæ in front.

Absorption of the papillæ, leaving the fornix rings intact.

Rupture of ectocyst through the orifices thus produced, causing renal colic with the passage of cysts twenty years ago.

Retention and growth of one cyst or scolex in the upper major calix.

Reconstruction of the original cyst from retained daughter cysts, scolices, or germinal layer elements. It is doubtful whether a new mother ectocyst was formed or not. Many of the small cysts appeared to be loose. Further egress to upper major calix would become shut off by tension of the wall of the new cyst growing there.

Undermining by cortex cyst of papillæ of the middle calix and eventual discharge of contents through the fornix ring and down the ureter, accounting for the recent attack.

The presence of the lower calix cyst is readily explained. In pelvic systole urine is forced back into all the calices as well as down the ureter. A scolex had at one time or another passed into the pericystic cavity of the upper calix cyst and been forced into the lower calix. If its hooklets were extruded, it might be entangled there while its fellows were swept out in the urine.

Several facts of interest emerge from the study of the case.

1. Hydatid cysts may flourish although part of the ectocyst is constantly bathed in urine.

2. They may gain sufficient nutriment by osmosis from a loose adherence to the epithelial wall of a calix, getting the necessary serum elements from the blood and excluding toxic ones from the urine.

3. The presence of a cyst in the calix does not necessarily cause any disturbance of the function of the papillæ or surrounding cortex.

4. The remarkable toleration of the calix to the foreign body.

Probably the vague abdominal pains for which the patient was operated on were due to mild calix colic, but for the last ten years even this symptom had failed.

MICROSCOPICAL EXAMINATION (*Fig.* 12).—Sections were cut through the wall of each cyst. In the upper calix cyst most of the transitional epithelium was missing, though strangely enough part of the adventitia of the cortex cyst had been epithelialized, presumably by continuity through the communicating orifice. The epithelial lining and muscular layer of the

FIG. 12.—*Case* 1. Low-power view of section through the wall of the lower calix cyst. The transitional epithelium lining the calix is intact. Two sections of a cut artery are seen in the middle of the pericyst

lower calix was preserved. The following layers could be demonstrated in all three specimens from within out: (1) Shreds of hyaline laminated membrane with adherent cells, as if from the germinal layer adhering to the inner surface of the cyst wall; (2) Epithelial layer missing in parts; (3) In the lower calix cyst the muscular layer of the lining membrane of the calix separated by loose areolar tissue from the rest of the pericyst; (4) Cellular fibrous tissue containing large blood-vessels; (5) Dense fibrous zone; (6) Advancing zone of fibroblasts with many plasma cells and eosinophils; (7) Kidney tubules and glomeruli.

Processes from the inner fibroblasts could be seen inserting themselves between tubules and the individual cells of the latter. These cells could still be detected singly and in groups in the midst of the advancing fibrous tissue.

Case 2.—Male, aged 32. Clerk.

HISTORY.—The patient lived as a child in the country. Referred for discharging lumbar and abdominal sinuses of twenty-three years' duration. From the age of 5 he had suffered from intermittent attacks of left renal pain. Four years later an abdominal tumour was discovered. Laparotomy was performed and an hydatid cyst of the lower pole of the left kidney cleaned out and marsupialized. Urine flowed from the resulting sinus for some years, but gradually the discharge became pure pus. Several later attempts made to remove the kidney

were unsuccessful and added a loin sinus to the existing abdominal one. Lately the patient has become weak and ill and subject to septic sores on arms and legs.

ON EXAMINATION (Jan. 28, 1924).—Cystoscopic and X-ray examination showed that the left ureter was occluded, the kidney functionless, that the sinus communicated with a pus cavity in the depths, and that the two sinuses together encircled the colon.

OPERATION.—A fibrolipomatous mass containing a cavity corresponding to the original renal pelvis was removed by sharp dissection. This mass was all that was left of the kidney.

SUBSEQUENT HISTORY.—A fæcal fistula due to local damage to the colon's blood-supply took nine months to heal. Eleven years after the operation the patient remains well with firm scars and no evidence of hydatid disease.

Case 3.—Female, aged 18. Single.

HISTORY.—The patient was a country girl and had had no previous symptoms. She came to Wellington to be confined. Primipara. Normal labour. On the fifth day of the puerperium she developed severe radiating pain starting in the right loin. Urine became turbid and some membranous material was passed by the urethra. This proved on microscopical examination to be hydatid membrane.

ON EXAMINATION (March, 1924).—On examination twenty-four hours after she had passed the membrane, the urine was clear and contained no hooklets or scolices. The only physical sign was some tenderness on pressure in the right costovertebral angle.

DIAGNOSIS.—Hydatid cyst of the right kidney. The patient was asked to report for detailed examination after she was up and about. However, she left the maternity home, got married, and was not seen again.

Case 4.—Male, aged 52, labourer. Admitted to Wellington Hospital, Nov. 5, 1935.

HISTORY.—Chronic cough for eighteen months, with loss of weight. Has never had urinary symptoms.

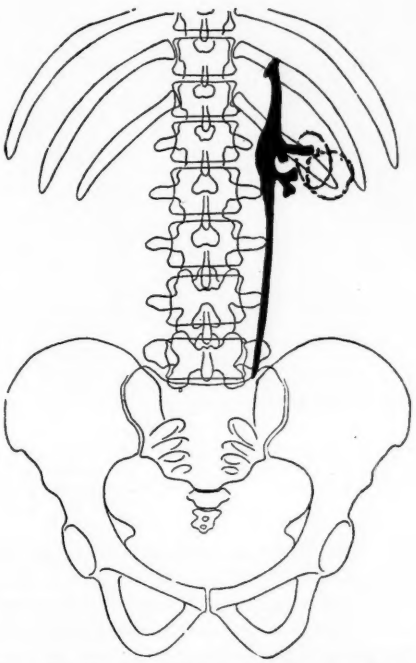


FIG. 13.—*Case* 4. Diagram of the pyelogram.

ON EXAMINATION.—Diminished breathing over the lower lobe of the right lung. No dullness. Breathing shallow but vesicular. Chest movements poor. No abdominal findings except that on deep inspiration, a tumour the size of a small orange could be felt in the left hypochondrium. It had the characteristic feel of a kidney tumour and moved freely on respiration.

X-ray Examination.—There was no evidence of hydatid cyst in the lungs, the radiograph tending to confirm the provisional diagnosis of carcinoma of the lung. Behind the twelfth rib on the left side at the outer border of the kidney shadow appeared two intersecting ring shadows typical of calcified hydatid pericyst.

Urine.—Trace of albumin, a few erythrocytes, pus cells, and granular casts. No hydatid elements.

Special Hydatid Tests.—Casoni, positive; Ghedini (complement-fixation), strongly positive. Eosinophils, 2 per cent.

Pyelogram of the Left Kidney (*Fig.* 13).—The lower major calix was normal. The upper and middle major calices formed a right angle with each other, giving the impression that

FIG. 14.—*Case* 4. Posterior aspect of the left kidney showing the two cysts.

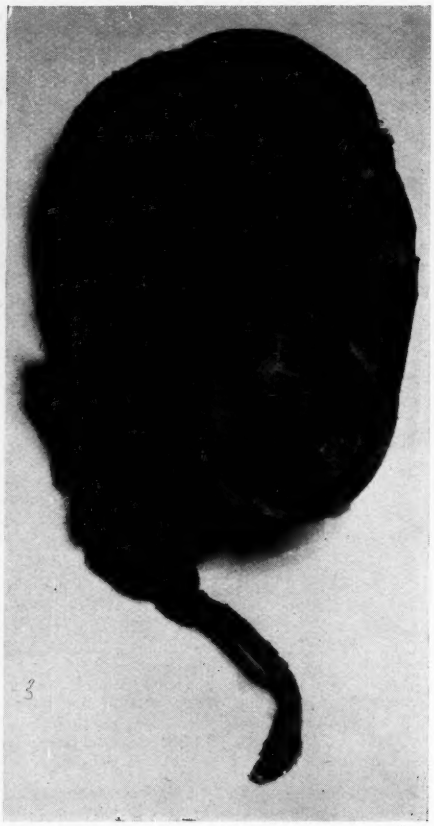


FIG. 15.—*Case* 4. Anterior aspect of the same kidney. All the cyst contents have been removed. Windows have been cut in both cyst walls but have been replaced in position.

a cyst was prising them apart. They were somewhat dilated and lengthened and the end of the middle major calix almost reached the circumference of the outer of the two ring shadows.

DIAGNOSIS.—As no hydatid material was found in the sputum at any time and there was no other evidence of hydatid disease of the lung, the hydatid cysts were looked on as an incidental finding. It was in doubt whether the cysts were in the spleen or kidney, but while the pyelogram was not pathognomonic a provisional diagnosis was made of a calcified cyst of the outer border and another cyst of the upper part of the left kidney.

COURSE.—The patient died on Nov. 21, 1935, from terminal pneumonic symptoms.

AUTOPSY.—The three important points which were revealed were: (1) Death was due to cancer of the lung; (2) There were two primary cortical hydatid cysts in the left kidney; (3) There was no evidence of hydatid infestation elsewhere in the body.

THE SPECIMEN (*Figs.* 14–16).—The kidney and the cysts together formed an almost quadrilateral mass 5 in. long by 4 in. wide. One large cyst oval in shape and measuring 8 × 5 × 5 cm. occupied the upper and lateral part of the kidney, reaching rather more than half-way down. It had displaced the upper major calix medially, and the middle major calix inferiorly, so that these two channels formed a right angle with each other as seen in the pyelogram. The lower cyst was more irregular in shape. Below, it formed a circular cavity attached by a wide pedicle to the lower part of the posterior border. The cyst had grown upwards, forming a tapering projection which lay lateral to the large upper cyst. Its medial wall was thus the lateral wall of the latter. Neither the endocystic nor the pericystic cavity of the upper cyst communicated with the kidney pelvis, but there was a small communication between the pericystic cavities of the two cysts about ½ cm. in diameter. The pericystic

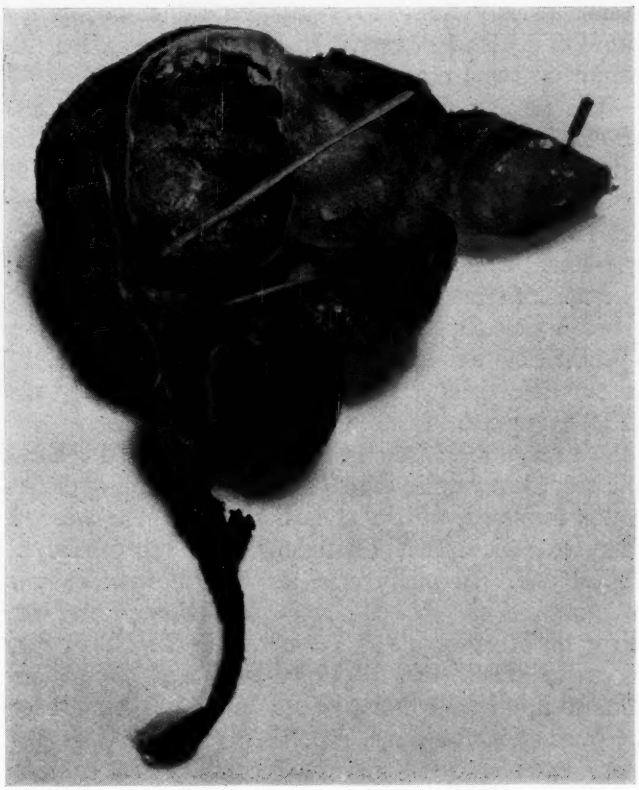


FIG. 16.—*Case* 4. The sectioned kidney—posterior half. The knife has passed precisely through the opening between the two pericystic cavities. The upper toothpick is laid across this opening. The lower shows the direction of the middle major calix thrust apart from the upper major calix so that the two form a right angle.

cavity of the lower cyst communicated freely with the middle major calix, but the contents of the endocystic cavity did not escape, the ectocyst being intact. The ectocyst of the upper cyst was also intact so that the contents of the two endocystic cavities did not mingle. When a pyelogram of the excised kidney was made, slight pressure resulted in the rupture of both ectocysts and the fluid penetrated in such a way that the daughter cysts which filled both cavities were clearly outlined.

Comment.—The presence of two separate primary cortical cysts is rare. The presence of blood-cells in the urine is an interesting and common finding in hydatid cysts of the pseudo-closed variety in spite of the total absence of urinary symptoms. The heavy calcification of the cyst walls, together with the size that the cysts had obtained, indicated that infestation had taken place many years before.

GENERAL OBSERVATIONS ON HYDATID DISEASE OF THE KIDNEY

In discussing the various aspects of the disease, reference will be made to illustrative cases from the literature by the insertion of the name of the author reporting the case in brackets, followed by the number of the case where the article referred to describes more than one. The location of the full report may be found by reference to the author's name in the bibliography.

History.—The first case in which hydatid cysts were passed in the urine was reported at the end of the seventeenth century by a London surgeon, Davies. Earlier in the same century Redi (1626–97), the Milan biologist, had established the parasitic nature of hydatid disease. In 1861 Béraud in his thesis initiated the systematic literature on renal echinococcosis.

Etiology.—The etiology is that of hydatid disease in general. The industry of sheep-raising on the one hand, and insanitary conditions of life on the other, contribute chiefly to its incidence and spread. The former factor prevails in Australia, New Zealand, and the Argentine, the latter notably in Greece and Dalmatia (Toole, Račić).

Pathology.—Renal cysts may be classified pathologically as: (1) *Primary*: derived from a hexacanth embryo. (2) *Secondary*: from a scolex, piece of detached germinal layer, brood capsule, or daughter cyst from some other part of the host. (3) *Invasion*: penetrating the kidney from an adjacent organ. Secondary cysts may be further subdivided into: (*a*) Embolic—conveyed by the blood-stream; (*b*) Calix—sown after rupture of the primary cortical cyst.

1. Unlike the liver, the kidney is almost invariably the site of one primary cyst only. Račić records a case in which a kidney containing eight cysts, each with daughter cysts, was removed, with a clinical cure. These were, however, almost certainly secondary embolic cysts. Frequently there are calix cysts in addition to the primary cortical one. In many cases at post-mortem examination the renal cyst is the only one in the body. However, others, dead or alive, may be found in the liver or other organs. Toole found the kidney cyst the only one in 42 out of 46 cases, Nicaise in 416 out of 449 cases. Some of these, however, were purely clinical findings. Other cysts may well have been missed, more especially in the pre-radiographic era.

2*a*. Secondary embolic cysts are usually multiple and accompanied by cysts elsewhere. Even in the latter case one kidney may be spared (Peacock and Wale Hicks), but both are more commonly involved (Welsh and Chapman, Kinyon, Dónovan). While secondary cysts from a ruptured liver or spleen may attach themselves to the peritoneal surface of the kidney (Allaben), there is no authentic record of invasion of the organ. Rupture of a cyst into the heart cavity is the usual cause of metastatic spread. Bocker found in one such case that the left renal artery was blocked by a daughter cyst. Liver cysts may, however, be associated with multiple secondary cysts of the kidney, the lungs remaining free.

Secondary renal cysts are rare compared with primary (25 out of 474 cases in Nicaise's series). They may contain daughter cysts, rupture, and behave exactly like the primary ones.

2*b*. Calix cysts are well illustrated in *Case* 1. They are always secondary to cortical cysts. Primary cysts of the renal pelvis are unknown. Of the two

mentioned by Nicaise, one (Bland-Sutton) was an error in translation, the other an unverified personal communication. Calix cysts as a rule are small and discoverable only by section of the excised kidney. Only one has been hitherto recognized as such (Willans and Stewart). Other illustrations leave little doubt that the secondary cysts were in one of the calices (Lutz, Livermore, Vila, and probably Filienz and Melen). In cases of complete kidney destruction the pelvis may be detected as a separate slit-like cavity or it may contain the cyst. The latter condition is probably the end-result of a calix cyst.

3. Invasion cysts in which liver or spleen as well as kidney are involved (Craig and Lee-Brown, *Cases* 9 and 10, E. L. Young, *Case* 2) leave some doubt as to the organ primarily affected. The pyelographic evidence is in favour of the kidney as a rule. Alivisatos, however, reports an undoubted case of rupture of a liver cyst into the right renal pelvis.

Echinococcus Alveolaris.—Only two kidney cases of this curious variant have been reported, those of Sabolotnow in 1867 and of Hahine in 1928. In Hahine's case the nodule in the kidney was secondary to one in the liver. It is still maintained by Posselt, of Innsbruck, that this variety constitutes a separate species. Evidence is accumulating against it. Dévé recently abandoned the dualist theory in an article entitled "Transitional Forms between Hydatid Echinococcus and Alveolaris" in which Dew's Australian case of *E. alveolaris* was cited. Posselt, however, returned to the attack with the uncompromising title, "There are no Transitional Forms". There the matter rests, and in any case the alveolar form in the kidney is a mere curiosity and requires no further mention.

Side Affected: Age and Sex Incidence.—In the larger series of cases both kidneys are equally affected and bilateral cysts are rare. Nicaise found 197 with the right kidney involved, 185 with the left, and 12 bilateral. The sexes are equally vulnerable.

Most cases are infected in childhood; the third to the fifth decades are the commonest periods for the development of symptoms. In the reports the youngest patient operated on was a child of 2 years (Schepel).

The percentage of kidney cases to all hydatid ones ranges from below 1 per cent in Argentine statistics to 4·65 per cent in Račić's series in Dalmatia. Compared with other surgical diseases of the kidney echinococcosis is very rare. Many urologists with large practices have never seen a case. The largest personal experience was that of Craig and Lee-Brown.

Site of Implantation.—The commonest site is the cortex of the anterior surface of the upper and lower poles. Cysts of the posterior surface as in *Case* 1 are rare. The cyst may arise in the loose tissue of the sinus and be adherent to the pelvis of the kidney (Bland-Sutton, Guiteras, Spurr, Rowlands, Keyes and Busch, Vedenski, 4); in the middle portion, bisecting the kidney and leaving the two poles intact (Fillenz, Račić, 10, Vedenski, 3); or it may occupy the whole kidney, only a minute nodule of renal substance remaining (Staveley, Marsh, Russell and Kilbane, Meltzer, Craig and Lee-Brown, 10, Leighton and Lewis).

The cyst may be very superficial, almost pedunculated (Erdélyi, Woodman, 2, Stark, Flynn, Geyman, Bressot). It may be so small as to defy detection until the kidney is completely split open (Craig and Lee-Brown, 16) or so large as to fill most of the abdomen (E. L. Young, 2, Craig and Lee-Brown, 2, Vedenski, 5, and Marsh). In Marsh's case the cyst measured 29½ by 13 in. and weighed 10 lb.

Development and Ultimate Fate of Parasite.—In the kidney, as elsewhere, the hydatid cyst develops very slowly, though growth may be stimulated by trauma or pregnancy. Expansion is centrifugal but the centre point of the sphere is not static. As a rule the line of least resistance is followed, but in the effort to preserve its circular contour the cyst may burrow into the spleen (Craig and Lee-Brown, 10), into the liver (Craig and Lee-Brown, 9, E. L. Young, 1), or the tail of the pancreas (Perrin). A fibro-cellular proliferation—the pericyst—acts as advance guard, the fibroblasts sending their processes between individual tubules, then between the cells, which appear as islands in a flood before being finally overwhelmed and absorbed. The outer circumference of the cyst, once it has reached and passed the kidney capsule, makes rapid progress into the perirenal tissues or bulges into the peritoneal cavity. Simultaneously the inner circumference may reach the calix wall, which offers resistance. The papillæ, being of soft tissue, are first absorbed. The pericyst may remain intact, or, left in the air, lose blood-supply and necrose. In the latter case the cyst becomes one of the pseudo-closed group and the ectocyst, though still intact, presents directly into the lumen of the calix. It may, however, hold for a long time, as the inner tube of a motor tyre may hold in spite of a small defect in the outer cover. The communication of the pericystic cavity with the calix may set up irritation and produce calix colic. Finally, owing to extra pressure, trauma, or strain, the ectocyst itself bursts and the contents pass through the fornix ring into the calix and down the ureter. This appears to be the sequence of events of the rupture of the cyst into the pelvis. Sometimes an escaped daughter cyst or scolex takes root in the calix wall and eventually by its growth and tension blocks the hole through which it came as a sail drawn under a ship's hull stops a leak. The scolex may be conveyed, by the current of urine, to a distant calix and grow there. The primary cortex cyst continues to grow and may eventually burst into yet another calix (*see Fig.* 11). Trauma may cause rupture of the ectocyst (Ivanov, Grove), or pregnancy or labour pains may determine the final break (*Case* 2, above, Vedenski, 3, Cowan, Flynn, Craig and Lee-Brown, 5). Even simple abdominal palpation may produce the same result (Mensikov).

The escaped cysts and hydatid debris may lie in the pelvis and ureter and cause no discomfort, or there may be violent renal colic, eventual dilatation of the ureter, and even hydronephrosis from obstruction of the duct. The cysts may stay in the bladder, causing strangury, difficulty, and retention.

The hydatid may die of old age or lack of nutriment. There is a ring of large blood-vessels in the pericyst, a striking microscopical feature (*see Fig.* 12). Thus ample serum is brought to the hyaline laminated membrane from which the parasite can select its food supplies. Calcification hinders the osmotic process. The animal feels the urge for more defensive propagation. Daughter cysts are born in large numbers and even granddaughter cysts (Farkas). Tension may become low and growth in abeyance or spasmodic. The whole tribe may perish and be buried like criminals in lime, or sepsis may supervene with unpleasant results both for guest and unwilling host. Low-grade life may, however, be maintained even though the pericyst is heavily calcified. Daughter cysts resist sepsis for a considerable time (Bird). The cysts may be sterile, or, though univesicular, contain brood capsules and scolices (Schmidt, Vedenski, 4 and 5). Far more frequently they are full of daughter cysts at the age period when the patient usually becomes sensible of unpleasant symptoms. Propagation is by endogenous budding, but the Russian

workers Massalitinov, Napalkov, and Keropian consider that reproductive elements in the pericyst are the chief cause of recurrence after operation. It is true that in confined spaces, as in bone, exogenous budding takes place by herniation (Dew), but it seems more probable that the fertile causes of recurrence are the pieces of the ectocyst that adhere to the pericyst even when the former appears to be loose and easily removable (Kaïris).

Complications.—The expanding pericyst may become incorporated with the stomach, spleen, pancreas, colon, liver, duodenum, or vena cava. The adrenals may become inseparably fused. Such a welding of tissues becomes a danger at operation, chiefly where the surgeon confuses it with ordinary adhesion and attempts to separate the inseparable. There is no reason why a large upper pole cyst should not rupture into the bronchial system. The five reported cases are, however, unconvincing (Nicaise 2 cases, Heer, Nicolich, and Christie and Fawcitt). In one of these at least a cyst was found subsequently in the lung. Empyema by lymphatic spread of sepsis may occur, or from bacteria accumulating outside the laminated membrane like debris on a filter paper. Abdominal cysts may rupture into the peritoneum. Such a case, as yet unpublished, exists in the Registry of Hydatids kept by Sir Louis Barnett for the Royal College of Surgeons of Australasia. Another ancient case is reported by Nicaise. I can find but one solitary report of rupture into the stomach, a fatal complication (Nicaise), and three into the colon, with amelioration of symptoms. One of these three, however (Parlavecchio), proved to be a pararenal cyst associated with another abdominal one. It was the latter that discharged into the colon. Sudden death from rupture into the vena cava such as has occurred with liver cysts (Pitts, K. D. Fairley) has not been reported. Indeed, the opening of a kidney cyst into any other viscus than the kidney pelvis is so rare that the possibility may almost be discounted in estimating the prognosis. Embolic spread from the kidney is also so rare as to be negligible.

Complications within the urinary tract include hydronephrosis, pyonephrosis, lowering of kidney capacity on both sides from difficulty in micturition or chronic retention due to accumulation of membrane in the bladder, with or without bilateral ascending pyelonephritis. All are rare and mostly due to sepsis. The ureter on the affected side may become completely occluded (*Case* 2 above, Haynes, Lutz). In the four recorded cases of solitary kidney with hydatid infestation, kidney function was well maintained (Blackburne, Houzel, Nicaise, 2). Multiple cysts may kill from uræmia through destruction of kidney substance (Kinyon, Donovan) or by an associated abdominal cyst or cysts blocking the ureter of the unaffected kidney. A perinephric abscess may develop and point in the loin.

Associated Pathology.—Calculus associated with hydatid cyst is rare even in such countries as Dalmatia where both conditions are common. Nicaise in his large series reports only twelve cases and includes stones in the bladder. Other cases are those of Vedenski, 4, Craig and Lee-Brown, 16, Blackburne (in a solitary kidney), Castaño and Astraldi, and an unpublished case in the R.A.C.S. Registry. I can find no other examples. Other instances of associated pathology are: malignant growth (Erdélyi), floating kidney (Eberle, Celowski), ectopic kidney (Juvara), adenoma (Galvez and Garate), and carcinoma of the bladder (Thomas). Ferruccio had a case of associated Addison's disease, but it is not clear whether this was due to adrenal involvement. Cancer may develop in a marsupialized cyst (Bonamy).

Symptomatology.—No symptoms at all may appear for many years, and the patient may be quite ignorant of the existence of the cyst till the day of his death. Closed cysts may be found accidentally in perfectly well individuals. The parasite may produce unexplained anaphylactic phenomena such as urticaria or obscure pressure symptoms. Those that grow towards the abdomen cause indigestion, pain, and fullness after meals by encroaching on the stomach, or diarrhœa, constipation, or even complete obstruction by pressure on the colon. In rare cases a gradual loss of strength, weight, and energy with ultimate symptoms of uræmia may result in death and the cause be discovered only at autopsy.

Pseudo-closed cysts cause the intermittent localized pains of calix colic and urinary symptoms of frequency and hæmaturia, accompanied by painful micturition and urgency if sepsis has supervened. All symptoms may be initiated or augmented by trauma, pregnancy, or labour.

The rupture of the ectocyst may be heralded by a sudden excruciating attack of pain in the loin or upper abdomen with extreme tenderness and rigidity of the abdominal muscles. Accentuation of the symptoms may be due to anaphylaxis added to spasm of the renal pelvis and ureter. Such cases have more than once been subjected to immediate laparotomy with a view to the repair of a supposed perforated gastric or duodenal ulcer. The very first symptom may be retention of urine due to an accumulation of membrane in the bladder. The temperature may be raised and the pulse rapid even when there is no sepsis, especially during or following attacks of renal colic. In more than half the cases a mild attack of colic is followed at the end of twenty-four or forty-eight hours by some difficulty in micturition and the eventual passage of membrane or daughter cysts.

The attack may never recur or may be repeated at intervals of a month, a year, or even many years. The onset of severe sepsis modifies the subsequent symptoms. In its absence the renal colic may become less severe with each attack owing to dilatation of the ureter, whereas the difficulty in emptying the bladder may be greater owing to the increasing abundance of hydatid material which reaches it. Between attacks the general health is, as a rule, excellent, and there is complete freedom from symptoms. The onset of infection may convert the intermittent loin pain into a constant nagging ache and there may be superadded strangury, frequency, scalding, and later the evidence of bilateral pyelonephritis in loss of appetite and general debility.

Diagnosis.—In open cases diagnosis is made from the examination of the material passed. If this is only 'hydatid sand' or brood capsules, the case may be considered merely a 'gravel' one, and the patient has perhaps to wait some time before a microscopical examination of the sediment discloses hooklets and scolices (Huffman). Calcified cysts may produce solid debris which when passed is mistaken for calculus (Croveri and Placeo). Retrovesical hydatid cysts fully reported on by Deeming very rarely rupture spontaneously into the bladder. I have found five records only (Nicaise, Fairbanks, Compan, Craig and Lee-Brown, Oritz and Garcia). An additional case in which one of a group of multiple secondary cysts opened into the bladder is on record in the R.A.C.S. Registry. Confusion of such cases with renal echinococcosis is most likely when the symptoms of the latter are confined to bladder difficulties with no renal pain or colic (Vedenski, 1 and 2). Cystoscopy with pyelography should clear up the diagnosis. It was the fortunate lot of two surgeons, Blum and Pereschevkin, actually to see a daughter

cyst extrude itself from the ureter. Craig was able to pass a cystoscope into a retro-vesical cyst. The bare possibility of a liver cyst rupturing into a kidney pelvis (Alivisatos) must be remembered.

Pseudo-closed cysts are demonstrated by typical pyelograms. Closed cysts are the most difficult. Clinically they appear as tumours in the hypochondrium or free and mobile in the abdominal cavity (Račić, 10). If the muscles are rigid, such a tumour may be missed during an attack of renal colic. An hydatid tumour feels solid. If fluctuation is detected some other pathology is more likely (Bird). Hydatid 'thrill' ('Schwirren', 'frémissement'), well described by Barnet, is so rare as to be valueless in most cases. The conditions required are a few large daughter cysts, fluid under low tension, a thin abdominal wall, and contact of a daughter cyst with that wall of the mother cyst which lies under the examining hand. The sensation is of a delicate metallic spring vibrating, accompanied by a sonorous resonance on auscultation. The 'thrill' in renal cases was detected in the cases of Burburg, Peyrot, Spurr, and Christie and Fawcitt.

The commonest errors in tumours appearing in the hypochondrium are to mistake the cyst on the left side for an enlarged spleen, especially where malaria is endemic, and on the right side for a liver cyst. In the former the special serum tests, with X-ray examination and pyelograms, usually clear up the diagnosis; in the latter serum tests are useless.

An upper pole cyst may produce high dullness at the lung base of the corresponding side and has to be distinguished from an intrathoracic condition or a splenic cyst as well as from a hydronephrosis. The greatest calamity in such cases is a diagnostic puncture.

Where there are no physical signs at all, routine urological examination may show deformity in the kidney pelvis and the serum tests eliminate other confusing renal conditions. In cases of neoplasm of the kidney, the ureter is often kinked, in cysts it maintains its contour (von der Becke). Any abnormality may, however, exist in the kidney and the serum tests be positive on account of hydatid in the liver.

X rays may show a density due to the high saline content of hydatid fluid or a typical curvilinear shadow of a calcified pericyst. In all types of case complete urological investigation usually gives fairly conclusive information, but the details of the cystoscopic and pyelographic findings are such that they can be treated effectively only at length, and in a separate paper. Abundant material for this purpose is now available from the cases of the last ten years.

Serum Tests.—The precipitin test, first put on an effective basis by the work of Fleig and Lisbonne, Welsh and Chapman, and others, is rarely used. The complement-fixation test, worked out by several separate workers, notably Ghedini, Imaz Apphatie, and Weinberg, between 1906 and 1908, is a laboratory proceeding requiring rigid conditions of technique to give its best results. The intradermal reaction of Casoni, taking into account both the immediate and delayed reactions, is simple, practical, and of great value. For the technique, interpretation, and use of these tests the reader is referred to the published writings of the workers in that field. Among the most prominent in the southern hemisphere are Imaz Apphatie, Nuñez, Dew, K. D. Fairley, N. H. Fairley, Kellaway, Williams, and Hercus.

Prognosis.—From the statistics available hydatid disease of the kidney appears on the whole to be relatively innocuous. Of the 474 cases of Nicaise 20 per cent were routine autopsy findings: 215 cases received no surgical treatment, and only

16 died as the direct or indirect result of the cyst. The first attack of colic and the passage of membrane may be the last in spite of an active parasite. Impairment of renal function to a dangerous level is rare even in old-standing cases, and the general health is seldom affected. Spontaneous cure results in many cases (Goinard), though symptomatic cure is probably more common than anatomical.

Prevention.—Preventive measures for hydatid disease in general are retarded by the increased overhead cost added to the industry of wool and frozen meat production, and by ignorance, conservatism, and poverty in the older countries. They consist of the establishment of bureaux and registries, public educational campaigns, and the elimination of the tapeworm in dogs by cooking of all offal fed to them, strict regulations to keep them out of the neighbourhood of abattoirs, and the regular administration of vermifuges. Progress has been made, but results must necessarily be slow in appearing, and indeed will not be fully apparent for a generation, as infection is in childhood and manifestation of symptoms in middle life.

Treatment.—Treatment is mainly surgical or expectant. Turpentine has been used to assist elimination of the daughter cysts (Mackey). No intravenous therapy is effective. Nephrectomy was introduced by Bockel to replace the universal method of marsupialization in 1887. Intervention must be governed by two considerations. In the first place the disease is a benign one, and a cyst once communicating with the pelvis, as a rule, ceases to grow because the level of tension required in the fluid for this purpose re-opens the safety-valve. This rule is subject to modification where a tense calix cyst prevents the re-using of the original portal of communication, as in *Case* 1 (*see Fig.* 11). In the second place radical operation is difficult owing to dense adhesions and is not to be lightly undertaken. As a rule no operation should be performed :—

1. After a first attack in which cysts are passed in the urine and investigation shows that there is only a small cyst and the kidney function is good on both sides. The danger of complications is small and subsequent intervention may be undertaken should disabling symptoms or septic complications occur.

2. If the other kidney is absent, infantile, or functioning badly from any cause, and no serious disability is caused by the hydatid. In the best hands any operation on a difficult kidney may lead to damage demanding nephrectomy and the risk is greater than leaving the parasite. Houzel unknowingly removed a solitary kidney.

3. If the cyst is calcified and dead, unless passage of calcified fragments threatens damage to the urinary tract or is seriously disabling.

In most other cases nephrectomy, if possible, is the operation of choice if the other kidney is normal in function, for the following reasons :—

1. The mortality is low, the convalescence short, and health quickly restored.

2. Secondary calix cysts may exist, making recurrence inevitable if the kidney is left. They cannot be detected during any conservative operation.

3. An apparently closed kidney may really be of the pseudo-closed type, and with conservative operations there is not only the danger of recurrence but also of infecting the urinary tract.

4. In many cases the whole of the pericyst cannot be removed, and in these recurrence is common. If the kidney is left, this will manifest itself by the old symptoms of renal colic and the passage of membrane. When the kidney is removed the recurring cyst will cause no trouble, and in the course of many years will reach the surface in the loin where it can be easily dealt with.

When the cyst hardly invades the renal substance at all and has never caused urinary symptoms of any kind, partial nephrectomy with removal of the cyst may be undertaken, but if some of the pericyst must be left, recurrence is likely under any technique and it is better to remove the kidney.

All surgeons agree on nephrectomy if kidney function is markedly lowered, nearly the whole of the organ is occupied by the cyst, or gross sepsis is present. The extreme conservative school go so far as to use partial resection even in open cases (Bengolea), or resect and repair part of the pelvic wall if the cyst is attached to it (Spurr). The only justification, it appears, for such procedures—and it has some validity—is that recurrence is not apparent, because the symptoms of it, owing to the slow growth of the cyst, may not appear in the patient's or the surgeon's lifetime.

In many cases the pericyst is so welded to the surrounding structures that it is quite unjustifiable to endeavour to remove it all. Attempts to do so have led to immediate death from hæmorrhage (Fulton), to tearing of a mesenteric artery with a fatal result (Mensikov), and to wounding the pedicle of the spleen with compulsory splenectomy (Perrin). In such cases the cyst may be cleaned out, formalinized, and left with primary suture of the parietes; or Račić's aseptic marsupialization method may be used. This consists in filling the cavity with antiseptic gauze and not re-dressing till the tenth day. Tube drainage should only be used for pus cases or those in which a pseudo-closed cyst is suspected and it is impossible to remove the kidney. In the latter there will be a urinary discharge following the operation. Marsupialization prevents recurrence by the prolonged sepsis killing both the parasite and the kidney, as in *Case* 2 reported above. Vila had to perform nephrectomy on three of his cases for recurrence after conservative operations. It is, however, better to risk recurrence than subject the patient to tube drainage or ordinary marsupialization in non-septic cases.

The ureter should be severed well down to prevent recurrent cysts discharging through it, but complete ureterectomy, even though loose cysts may be left as in *Case* 1 and in Willans and Stewarts's case, is unwise. Fresh planes for recurrence are opened up; an operation, in any case difficult, is prolonged; and there is no recorded instance where a cyst has recurred in the ureteral stump.

Operation should be by the lumbar route, with the necessary forward extension and rib resection in difficult cases. The transperitoneal route handicaps the surgeon because there is no direct access for the sharp dissection of adhesions, and apart from other cogent considerations the mortality is higher (32 per cent against 8·5 per cent in the statistics of E. L. Young). In upper pole cases it is sometimes necessary to adopt the transcostodiaphragmatic route.

SUMMARY

1. A general description is given of the echinococcus and the route by which the embryo reaches the kidney, followed by an analysis of the published case reports of the last ten years.

2. Four personal cases are described.

3. The classification of the cysts of the kidney into closed, pseudo-closed, and open corresponds to certain cardinal symptoms.

4. Hydatid cysts of the kidney may be secondary, primary, or invasion.

5. The great majority are primary, but secondary calix cysts may be present and cannot be detected till the kidney has been excised and sectioned.

6. The prognosis is good, as renal cysts rarely rupture into neighbouring cavities or organs, with the exception of the kidney pelvis.

7. Diagnosis should be made in all open cysts and in a large per cent of closed ones by the use of urological methods, X rays, and the special serum tests.

8. The treatment where possible should, as a rule, be nephrectomy, but under certain conditions no operation should be done.

I have to thank Sir Louis Barnett for giving me access to the valuable cases in the Registry of Hydatids of the Royal Australasian College of Surgeons and for assistance in elucidating many points in the life history and habits of the parasite; also Dr. P. A. Treahy for his kindness in preparing the two coloured illustrations.

BIBLIOGRAPHY

The numbers in brackets indicate the number of case reports in the article referred to if more than one.

* Indicates that the case was a pararenal one.

ALIVISATOS, N., *Bull. et Mém. Soc. nat. de Chir.*, 1934, lx, 1112.
ALLABEN, J. E., *N. Amer. Pract.*, 1891, iii, 612, quoted by Kretschmer.
ANDRÉS, F. PÉREZ (2), *Clin. y. Lab.*, 1932, xx, 226.
ASCOLI, R., *Osp. maggiore*, 1932, xx, 407.
BABINI, R., *Gaz. deg. Osped.*, 1929, l, 581.
BARNETT, L. E., *Surg. Gynecol. and Obst.*, 1927, xlv, 148; *N.Z. Med. Jour*, 1921, xx, 277; 1934, xxxiii, 191; 1935, xxxiv, 1; *Jour. Coll. of Surg. of Australasia*, 1929, i, 351; *Austral. and N.Z. Jour. Surg.*, 1935, iv, 211.
BARNEY, J. D. (2), *Trans. Amer. Assoc. Genito-urin. Surgeons*, 1929, xxii, 19 and 30.
* BEDARIDA, N. V., *Minerva med.*, 1927, vii, 1330.
BENGOLEA, A. J., *Bol. y. Trab. d. l. Soc. d. Cir.*, 1926, x, 445.
BERAUD, E., *Thèse de Paris*, 1861.
BIRD, F. D., *Med. Jour. of Australia*, 1925, i, 258.
BLACKBURNE. Quoted by Nicaise.
BLAND-SUTTON, J., *Tumours Innocent and Malignant*, 5th ed., 652.
BLUM, V. (4), *Zeits. f. urol. Chir.*, 1933, xxxvii, 46.
BÖCKEL, *Etude sur les Kystes h. du Rein*, 1887. Paris: Alcan.
BÖCKER, A., *Inaug. Dissert.*, 1868. Berlin.
BONAMY, R., *Paris chir.*, 1910, ii, 100, quoted by Russell and Kilbane.
BOROSS, E., *Zeits. f. Urol.*, 1926, xx, 534.
BORRELL, J. H., and BARNES, J. M., *N.Y. State Jour. Med.*, 1933, xxxiii, 1390.
BRAILSFORD, J. F., *Brit. Med. Jour.*, 1931, i, 133.
BRESSOT, E., *Lyon chir.*, 1929, xxvi, 73.
BURBURG. Quoted by Nicaise.
BURFORD, C. E., *Trans. Amer. Assoc. Genito-urin. Surgeons*, 1929, xxii, 19 and 30.
CASONI, T., *Fol. clin. chim. et micro.*, 1911–12, iv, 5.
CASTAÑO, E., and ASTRALDI, A., *Rev. d. Especialid.*, 1926, i, 696.
ČELOWSKI. Quoted by Russell and Kilbane.
ČERVEŇANSKÝ, J., *Čas. lék. čes.*, 1932, lxi, 389.
CHRISTIE, G. W., and FAWCITT, R., *Brit. Med. Jour.*, 1934, i, 237.
COMPAN, V., *Arch. franco.-belg. de Chir.*, 1928, xxxi, 509.
COUNIOT, J., *Arch. d. Mal. d. Reins*, 1930, v, 192.
COWAN, A. F., *Jour. Obst. and Gynæcol. Brit. Emp.*, 1929, xxxvi, 592.
CRAIG, G., and LEE-BROWN, R. K. (16), *Surg. Gynecol. and Obst.*, 1928, xlvi, 668.
CROVERI, P., and PLACEO, F., *Boll. e Mem. Soc. piemontese di Chir.* 1932, ii, 149.
DAINELLI, M., *Ann. ital. di Chir.*, 1927, vi, 908.
DAVIES, Quoted by Nicaise.
DEEMING, C. L., *Jour. of Urol.*, 1923, x, 1.
DÉVÉ, F., *Thèse de Paris*, 1901; *Comptes rend. Soc. de Biol.*, 1913, lxxiv, 735; *Ann. d'Anat. pathol.*, 1933, x, 1155; *Austral. and N.Z. Jour. of Surg.*, 1934, iv, 99; *Comptes rend. Soc. de Biol.*, 1934, cxv, 954.

DEW, H. R., *Hydatid Disease*, 1928. Sydney: Aus. Pub. Co. *Med. Jour. of Australia Supp.*, 1924, i, 113, and 1925, i, 471; *Jour. Coll. of Surg. of Australasia*, 1929, i, 337; *Brit. Jour. Surg.*, 1930, xviii, 275; *Brit. Med. Jour.*, 1935, ii, 620.
DÓNOVAN, R. E., *Prensa méd. argent.*, 1931, xvii, 1460.
EBERLE, A., *Zentralb. f. Chir.*, 1911, xxxviii, 1656.
ERDÉLYI, J., *Forts. a. d. Geb. d. Röntgenstrahl.*, 1928, xxxviii, 858.
FAIRBANKS. Quoted by Deeming.
FAIRLEY, K. D., and associates, *Med. Jour. of Australia*, 1922, i, 346; 1923, ii, 27; 1929, ii, 320; *Jour. Coll. Surg. of Australasia*, 1930, ii, 317; *Austral. and N.Z. Jour. Surg.*, 1933, ii, 236.
FAIRLEY, N. H., *Med. Jour. of Austral.*, 1922, i, 341; *Quart. Jour. Med.* 1922, xv, 244.
FALIN, D. V., *Vestnik khir.*, 1934, xxxvi, 138.
FARKAS, I., *Zeits. f. Urol.*, 1926, xx, 534.
FERRUCCIO, B., *Gaz. deg. Osped.*, 1933, liv, 1307.
FILLENZ, K., *Zeits. f. Urol.*, 1926, xx, 65 and 96.
FINSEN, J., *Arch. gén. de Méd.*, 1869, xiii, 23 and 191; *Ugeskr. f. Læger*, 1867, iii, ser. 3, Nos. 5 and 8.
FLEIG and LISBONNE, *Comptes rend. Soc. de Biol.*, 1907, lxii, 1198.
FLYNN, R., *Austral. and N.Z. Jour. Surg.*, 1932, ii, 99.
FULTON, R. A. H., and BARNETT, L. E., *N.Z. Med. Jour.*, 1926, xxv, 317.
GALL, D. (66), *Ukraine nov. chir. arch.*, 1928, xvi, 564; *Zeits. f. urol. Chir.*, 1930, xxx, 84.
GALVEZ, I., and GARATE, F. O., *Rev. Assoc. méd. argent.*, 1932, xlvi, 622, 803, and 990.
GEYMAN, M. J., and CLARK, D. M., *Amer. Jour. Roentgenol.*, 1934, xxxi, 541.
GHEDINI, G., *Gaz. deg. Osped.*, 1906, xxvii, 1616; 1907, xxviii, 53.
GOINARD, P. (36), *Bull. méd. Paris*, 1930, xliv, 275.
GREENWAY, D. J., *Semana méd.*, 1922, ii, 761.
GROVE, H., *Bol. Soc. d. Cir. d. Chile*, 1927, v, 144.
GUITERAS, R., *Urology*, 1912, i, 496. New York: Appleton & Co.
GUTIERREZ, A., *Bol. y Trab. Soc. d. Cir.*, 1926, x, 442.
HAHINE, Z. D., *Russkaya klinika*, Moscow, 1928, ix, 315.
HAYNES, S., *Ann. of Surg.*, 1902, xxxvi, 95.
HEER, T. Quoted by Nicaise.
HERCUS, C. E., *N.Z. Med. Jour.*, 1926, xxv, 325.
HOUZEL, *Rev. d. Chir.*, Paris, 1898, 689 and 811.
HUFFMANN, L. F., *Jour. of Urol.*, 1926, xvi, 197.
HURLEY, V., *Austral. and N.Z. Jour. Surg.*, 1933, ii, 326.
IMAZ APPHATIE, I. L., *Rev. méd. lat.-amer.*, 1932, xvii, 1645.
IOANIDI, S. S., *Vrath Gaz.*, 1928, xxxii, 1113; *Zeits. f. urol. Chir.*, 1932, xxvii, 216.
IVANOV, N., *Med. univ.-klin. Voronezh.*; *Zeits. f. urol. Chir.*, 1928, xxvii, 216.
JEANNENEY, G., *Bordeaux chir.*, 1932, iii, 36.
JUVARA, E., *Bull. et Mém. Soc. de Chir.*, 1913, xxxix, 103.
KAÏRIS, Z., *Zeits. f. Urol.*, 1927, xxi, 677; *Zeits. f. urol. Chir.*, 1934, xxxix, 22.
KELLAWAY, C. H., and FAIRLEY, K. D., *Med. Jour. of Australia*, 1932, i, 340.
KEROPIAN, Monograph, 1930, Krasnodar, quoted by Račić.
KERSENTE, F., *Prat. méd. du Nord d. l'Afrique*, 1932, v, 622.
KEYES, W. C., and BUSCH, F. C., *Buffalo Med. Jour.*, 1891, xxxvi, 25, quoted by Kretschmer.
KINYON, J. J. Quoted by Kretschmer.
KIRWIN, T. J., *Jour. of Urol.*, 1926, xv, 273.
KRESTOVSKY, V., *Vestnik khir.*, 1927, x, 169.
KRETSCHMER, H. L., *Trans. Amer. Assoc. Genito-urin. Surgeons*, 1929, xxii, 19 and 30.
KRETSCHMER, H. L., (18) *Surg. Gynecol. and Obst.*, 1923, xxxvi, 196.
KRETSCHMER, H. L., and MOODY, A. M., *Surg. Gynecol. and Obst.*, 1914, xix, 766.
KÜSTER, E., *Die Chirurgie der Harnleiter*, 1896. Berlin. "Tierschmarotzer der Niere", *Deut. Chir.* (Bergman and v. Bruns), No. 52, 663.
LABOVITI, V., *Med. Welt*, 1932, vi, 743.
LANOS, J., *Bull. et Mém. Soc. de Chir.*, 1929, xxi, 628.
LEE-BROWN, R. K. (2), *Jour. of Urol.*, 1930, xxiii, 611.
LEIGHTON, W. E., and LEWIS, B., *Amer. Jour. Urol.*, 1912, viii, 71.
LIVERMORE, G. R., *Trans. Amer. Assoc. Genito-urin. Surgeons*, 1929, xxii, 19 and 30.
LUTZ, *Zeits. f. urol. Chir.*, 1929, xxvii, 165.
MACKEY, E., *Lancet*, 1905, ii, 601.
MACKEVNIN, N. E., *Vratch Gaz.*, 1927, xxxi, 260.
MANUJLOV, A. I., *Omsky Med. Jour.*, 1928, iii. 25.
* MARGARUCCI, O., *Arch. ital. di Chir.*, 1927, xviii, 447.
MARSH, J. O., *Cincinn. Lanc. and Observ.*, 1869, xli, 539, quoted by Kretschmer.

* MASSIMINO, A., and FINOCHIETTO, R., *Prensa méd. argent.*, 1930, xvi, 1576.
MASSALITINOV. Quoted by Račić.
MELEN, D. R., *Jour. Amer. Med. Assoc.*, 1925, lxxxiv, 1725.
MELTZER, M., *Ibid.* 1929, xcii, 1925; *Amer. Jour. Surg.*, 1929, vii, 418.
MENSIKOV, F., *Sibir. arch. teor. i klin. med.*, 1928, iii, 297; *Zeits. f. urol. Chir.*, 1929, xxvii, 404.
MOLLO, L., *Policlinico*, 1930, xxxvii, 557.
MORSON, A. C., *Proc. Roy Soc. Med.*, 1926–7, xx, pts. 1 and 2, 719.
NAPALKOV, N. I. Quoted by Račić.
NICAISE, *Soc. d. Chir.*, 1884. Quoted by Deeming.
NICAISE, V., *Thèse de Paris*, 1904–5; *Arch. gén. d. Chir.*, 1909, iii, 453; *Encyclop. franç. d'Urol.*, 1914, iii, 75.
NICOLICH, *Jour. d'Urol.*, 1912, ii, 843.
NÚÑEZ, J. O., *Prog. d. l. Clin.*, 1935, xliii, 31.
O'NEIL, R. F., *Trans. Amer. Assoc. Genito-urin. Surgeons*, 1929, xxii, 19 and 30.
ORITZ, A. F., and GARCIA, J. E., *Rev. med. latin-amer.* 1933, xviii, 529.
OULIÉ (4), *Bull. et Mém. Soc. nat. de Chir.*, 1928, liv, 718.
* PARLAVECCHIO, *Zentralb. f. Chir.*, 1909, xxxvi, 1702.
PEACOCK, T. B., and WALE HICKS, J., *Trans. Pathol. Soc. Lond.*, 1863–4, xv, 247.
PELS-LEUSDEN, F., *Zentralb. f. Chir.*, 1931, lviii, 2984.
PERESCHEVKIN. Quoted by Vedenski.
PERRIN, *Lyon chir.*, 1929, xxvi, 707.
PEYROT. Quoted by Nicaise.
PITTS, A. G., *N.Z. Med. Jour.*, 1919, xviii, 191.
PODDIGHE, A. (3), *Riforma med.*, 1929, i, 634.
POESSLT, A., *Frankf. Zeits. f. Pathol.*, 1934, xlvii, 194.
RAČIĆ, J. (10), *Zeits. f. urol. Chir.*, 1925, xvii, 163; *Zeits. f. Urol.*, 1931, xxv, 341; *Urologia*, 1934, i, 182; *Zeits. f. Urol.*, 1934, xxviii, 292.
RAVASINI, C., *Atti d. Soc. ital. d. Urol.*, 1929, 130.
* REGOLI, G., *Gazz. internaz. med.-chir.*, 1930, xxxviii, 293.
RÉVÉSZ, *Zeits. f. Urol.*, 1926, xx, 66.
ROCHE, E. H., and MACKENZIE, B., *N.Z. Med. Jour.*, 1935, xxxiv, 270.
ROWLANDS, R. P., *Guy's Hosp. Rep.*, 1929, lxxix, 117.
RUSSELL, J. I., and KILBANE, E. F., *Ann. of Surg.*, 1917, lxv, 585.
SABOLOTNOW. Quoted by Nicaise and Young and Davis.
SALLERAS, J., *Rev. d. Especialid.*, 1928, iii, 402.
SCHEPEL, J. A. C., *Nederl. Tijds. v. Geneesk.*, 1930, lxxiv, 5705.
SCHMIDT, L. E., *Trans. Amer. Assoc. Genito-urin. Surgeons*, 1915, x, 131.
SENANTE, V. FERRANDIZ, *Rev. d. Cir. d. Barcelona*, 1931, i, 388.
SIGINOLFI, P. (2), *Riv. radiol. e fis. med.*, 1930, Supp. No. 1, 129.
SILNIKOV, S., *Nov. chir. arch.*, 1928, xvi, 563; *Zeits. f. urol. Chir.*, 1929, xxviii, 403.
SORRENTINO, M., *Riforma med.*, 1930, xlvi, 783.
SPURR, R., *Rev. d. Especialid.*, 1928, iii, 402.
STARK, V. (2), *Vestnik khir.*, 1928, xiv, 92; *Zentralb. f. Chir.*, 1928, lv, 2888.
STAVELEY, A. L., *Montreal Med. Jour.*, 1890, xviii, 148, quoted by Kretschmer.
SURRACO, L. A., and associates (3), *Ann. de Fac. de Med.*, Montevideo, 1932, xvii, 668 and 818; *Rev. méd. latin.-amer.* 1931, xvi, 1453.
TARNOWSKY, G. DE, *Surg. Clin. N. Amer.*, 1927, vii, 783.
TAUBENSCHLAG, H., *Bol. y Trab. Soc. de Cir.*, 1925, ix, No. 4.
THOMAS, D., *Lancet*, 1879, i, 297; *Austral. Med. Jour.*, 1881, ii, 250, 344, 391, and 489; *Proc. Roy. Soc.*, London, 1885, xxxviii, 457.
TOOLE (46 renal, 10 pararenal), *Arch. f. klin. Chir.*, 1930, clix, 124.
VALDONI, P. (10), *Clin. chir.*, 1928, iv, 981.
VASILIU, SILVESTROVICI, and FRANKE, O., *Rev. san. mil.* 1934, xxxiii, 371 and 474.
VEDENSKI, D. A. (6), *Vestnik khir.*, 1928, xiii, 129; *Zeits. f. Urol.*, 1925, xxii, 694.
VILA, E. L. (8), *Rev. d. Cir. B. Aires*, 1929, viii, 158; *Bol. y. Trab. Soc. de Cir.*, 1928, xii, 785.
VON DER BECKE, A., *Rev. d. Especialid.*, 1928, iii, 402; *Rev. med. int. y tis.*, 1927, iii, 458; *Semana méd.*, 1927, ii, 1186; *Rev. d. Especialid.*, 1927, ii, 1029.
WEDENSKI, D. A., *see* Vedenski.
WEINBERG, M., *Ann. de l'Inst. Pasteur*, 1909, xxiii, 472.
WELSH, D. A., and CHAPMAN, H. G., *Lancet*, 1908, i, 1338; 1909, i, 1103.
WILLANS, E. T., and STEWART, M. J., *Brit. Jour. Surg.*, 1927, xiv, 667.
WOODMAN, G. S. (2), *Brit. Jour. Urol.*, 1931, iii, 299.
YOUNG, E. L. (3), *Cabot's Modern Urology*, 1924, 2nd ed., ii, 519. New York.
YOUNG, H. H. and DAVIS, D. M., *Young's Practice of Urology*, 1927, i, 358.

CONGENITAL DISLOCATION OF THE HEAD OF THE RADIUS

By BRYAN McFARLAND, Liverpool.

There is very little if any reference in the British literature to congenital dislocation of the head of the radius. It is barely mentioned in the surgical text-books and then only as part of another condition. Dislocation associated with congenital synostosis of the upper end of the radius and ulna is not a true dislocation, for the head is fixed by the fusion just below it, and may lie posterior to the ulna or anterior, or may in fact be non-existent. In some cases of birth palsy (Erb's type) a subluxation of the head of the radius develops as a result of muscle unbalance.

In the condition I am describing there is no fusion of the radius and ulna and no paralysis. The head of the radius is displaced forwards, is quite free from the ulna, and can be moved through a considerable range, but cannot be replaced in the normal position. This is significant because traumatic dislocation of the head of the radius, particularly if uncomplicated by fracture of the ulna, is reduced with ease and often spontaneously.

My attention was first attracted to the condition by being asked to treat a child who, having recently fallen, had apparently dislocated the head of his radius, and a house surgeon had twice unsuccessfully attempted reduction. I also failed by closed manipulation, and at open operation I found that the head of the radius was outside the capsule; that there was no orbicular ligament; that there was no lesser coronoid fossa; that the head of the radius was misshapen; and that the radius was too long, so that when one pushed the head against the capitellum, pressure was very great. I accordingly removed the head of the radius. I was able later to compare it with a dislocated head removed for mal-union of the ulna. The congenital head is small. Instead of being flattened as shown in *Fig.* 17, it is almost domed as in *Fig.* 18, with only a little dimple in place of the broad shallow capitellar depression (*see Fig.* 29).

Fig. 17

Fig. 18

About a year later another child was presented with the same story of injury. I regret to say that I did not recognize the significance of failure to obtain reduction by closed manipulation until I had again opened the elbow-joint. It was then immediately apparent to me that I was dealing with the same condition, and that it ought to be possible before operation to establish the congenital nature of the dislocation.

In radiographs the radius appears too long for the ulna, and the head of the radius appears ill-formed. There is a small area of ossification in the tissue overlying the head of the radius resembling the sesamoid type of calcification which sometimes occurs over the head of a congenitally dislocated femur. Subsequent cases showed that this 'sesamoid' was not constant. There is an anterior curve

of the posterior outline of the ulna, starting at the level of the coronoid fossa as in *Fig.* 19. It is sometimes slight but definitely contrary to the slightly posterior curve normally present at this level* (*Fig.* 20). A variation is also noticeable in

FIG. 19

FIG. 20

FIG. 21

FIG. 22

the anterior outline of the ulna. Instead of the backward sweep from the coronoid (*Fig.* 22) there is almost a straight line (*Fig.* 21). These features, together

FIG. 23.—*Case* 1. Congenital dislocation of the head of the radius. Note: (1) The radius is long; (2) The head is poorly formed; (3) The posterior outline of the ulna has a forward curve starting high up; (4) The anterior outline of the ulna has not the usual backward curve from the coronoid. (Cf. *Fig.* 25.)

FIG. 24.—*Case* 1. Congenital dislocation of the head of the radius. Six months after replacement of the head of the radius. The X-ray shows pressure atrophy and partial re-dislocation of the head. The shape of the upper end of the ulna is of course unchanged from that shown in *Fig.* 23.

* There is normally a faint anterior curve starting about the junction of the upper and middle thirds.

with the inability to reduce the head, point to a congenital dislocation rather than an acquired one.

When some time later a third case presented itself, it was easy to recognize

FIG. 25.—*Case* 2. Congenital dislocation of the head of the radius.

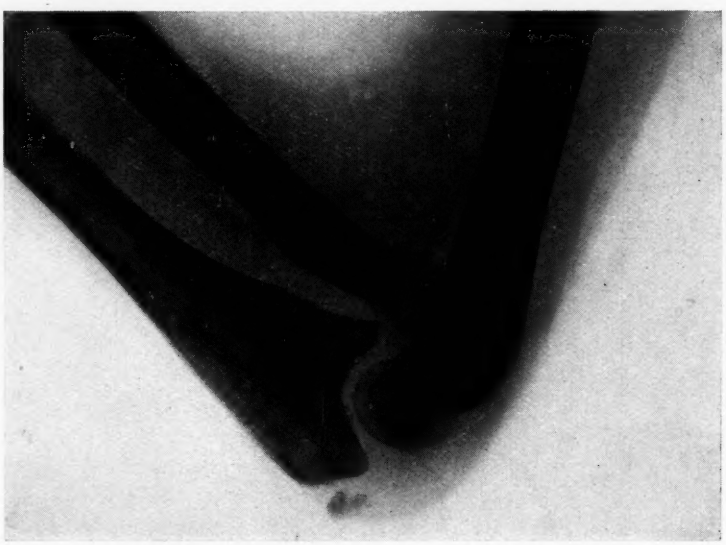


FIG. 26.—*Case* 3. Congenital dislocation of the head of the radius. Note the long radius and poor head and the shape of the ulna. *See Fig.* 27 for photograph of arm and *Fig.* 29 for scale drawing of head after removal.

the nature of the condition (*Fig.* 23). At operation the diagnosis was confirmed and on the suggestion of another surgeon I replaced the head of the radius. I did this against my inclination because I believed that a pressure atrophy (such as I

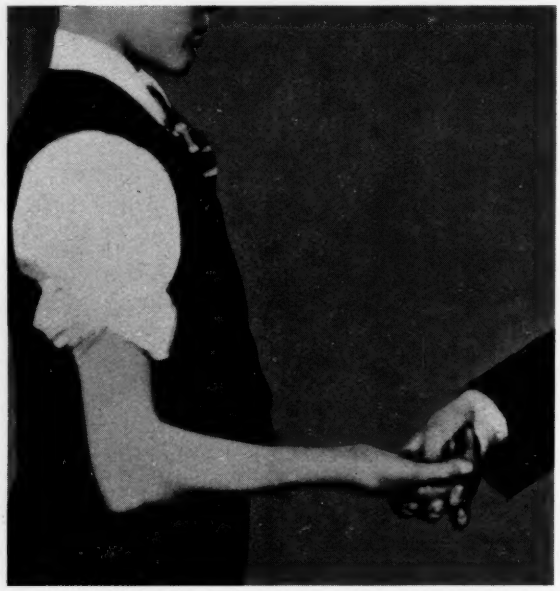


FIG. 27.—*Case* 3. Photograph of congenital dislocation of the head of the radius. Note posterior curve in outline of ulna. *See Fig.* 26 for X-ray, and *Fig.* 29 for scale drawing of head of radius after excision.

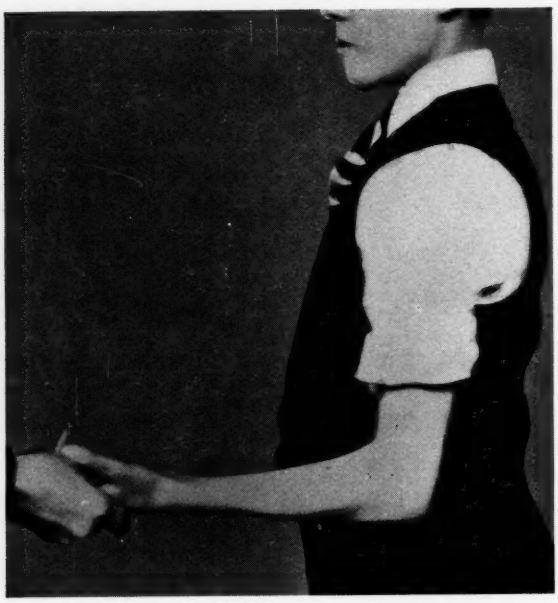


FIG. 28.—*Case* 3. Photograph of normal elbow. (Cf. *Fig.* 27.)

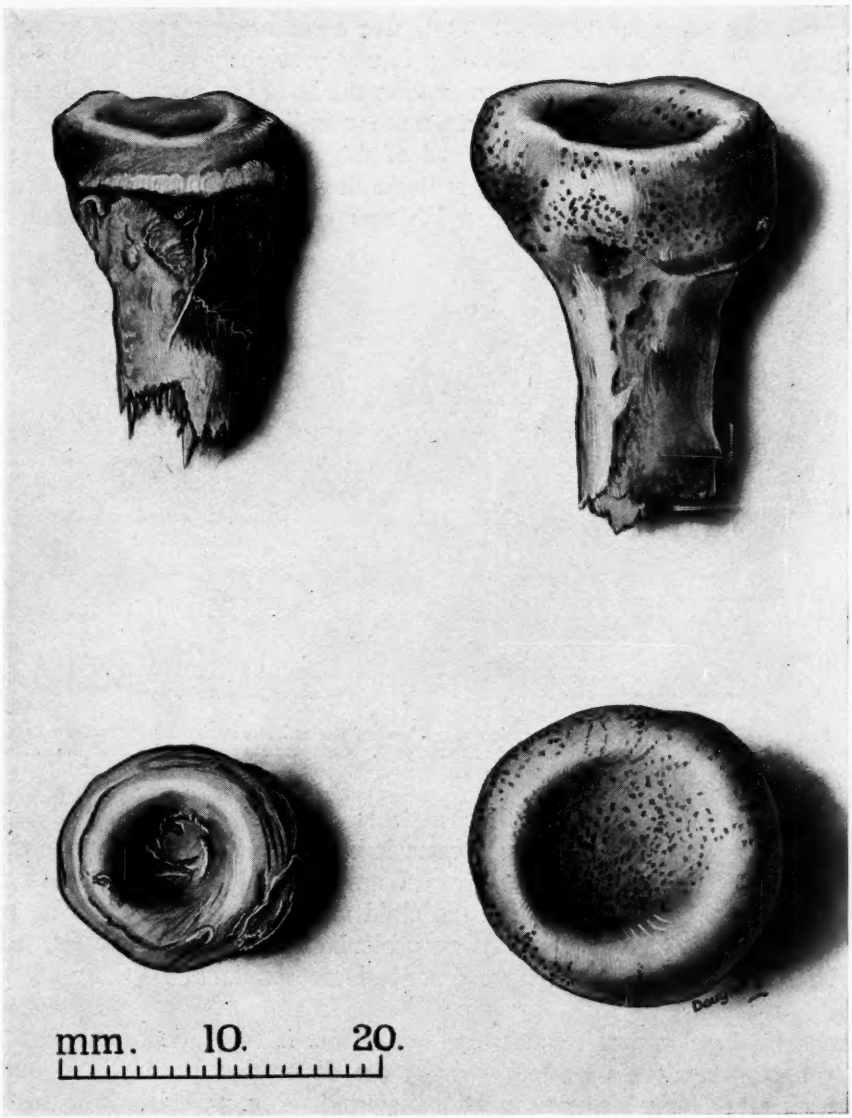


FIG. 29.—Scale drawings of dislocated heads of radius from patients of similar ages. On the left is the congenital type removed from *Case* 3 (*see Figs.* 26, 27). On the right is a normal head dislocated in association with mal-union of the ulna. (*See Fig.* 30, *Case* 4.)

have described in the head of the femur when an old-standing congenital dislocation is reduced) would occur and stiffness would result. This was so, and later I had to remove the head of the radius. (*Fig.* 24.)

It must not be assumed that it is always necessary to operate. Only for such patients as have loss of function should operation be undertaken.

Eleven children have been treated. In each case the dislocation was anterior. In none was the condition bilateral. Only five needed operation. (*Figs.* 23–37.)

Children usually attended following injury previous to which misplacement had been unnoticed. On clinical examination the head of the radius was felt to be prominent in the antecubital fossa near the mid-line and irreplaceable. There was, of course, a hollow where the head of the radius should be, and the slight anterior ulnar curve noticeable in the radiograph was clinically appreciable (*see Figs.* 27, 33). In over half the cases function was unimpaired. In no instance was

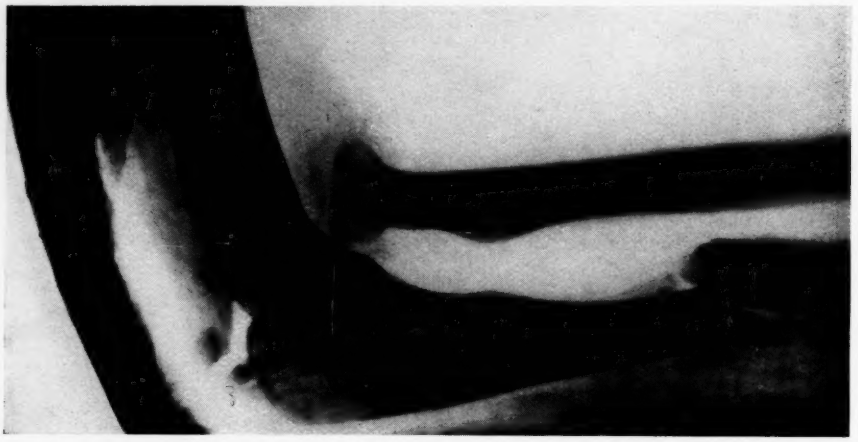


FIG. 30.—*Case* 4. Traumatic dislocation of the head of the radius associated with mal-union of the ulna. *See Fig.* 29 for scale drawing of head after removal. Compare shape of head with *Figs.* 23, 26, etc. Also note normal curves of ulna.

extension or rotation affected. Five patients suffered from continual or intermittent blocking of flexion. In such cases it is a simple matter to excise the head of the radius. For reasons I have given I am opposed to attempting to replace the head. For successful reduction the child must be young, preliminary traction must be employed, and at operation an orbicular ligament must be formed.

The X-ray appearances I have described are shown in *Figs.* 23–26, 31, 32, 36, 37.

Congenital dislocation has an added importance in that a doctor may be blamed for not having efficiently treated an arm, the dislocation having been unnoticed before the injury for which treatment was started. *Fig.* 35 brings out this aspect. The boy had sustained a fracture of the ulna for which he had been treated by a general practitioner. His parents complained of: (1) A lump on the back of the arm; (2) The lump in front of the elbow interfering with function. It was seen from the radiograph that the alinement of the ulna was adequate and union was satisfactory. I was able to establish that the head of this radius had been dislocated before the accident, and was in fact a congenital dislocation. The lump on the back of the arm was of course merely callus and gradually disappeared.

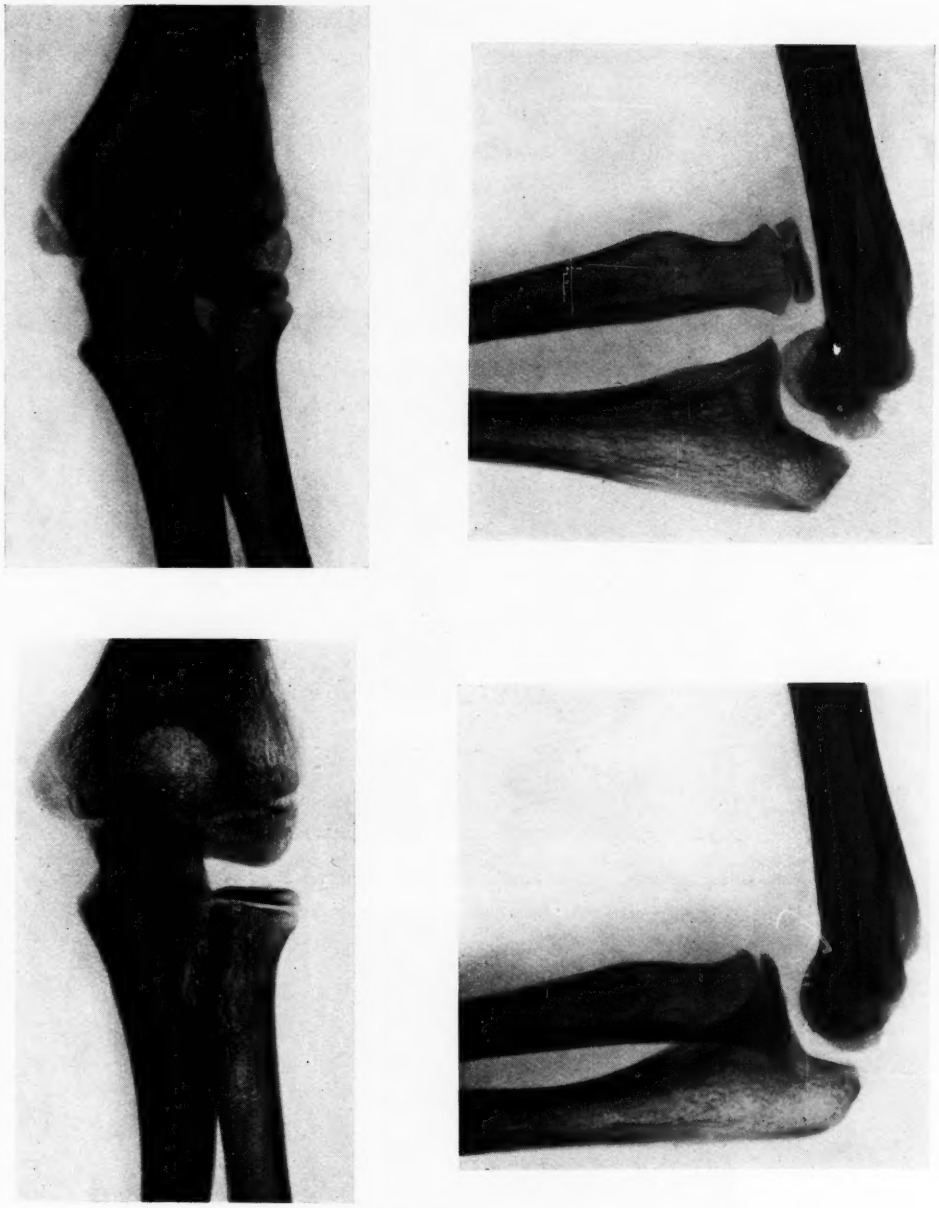


FIG. 31.—*Case* 5. The upper radiographs show the dislocated right joint. Those below, showing the normal left elbow, have been reversed in printing to make comparison easier. Note the changes in the radius and ulna and especially the excessive length of the radius.

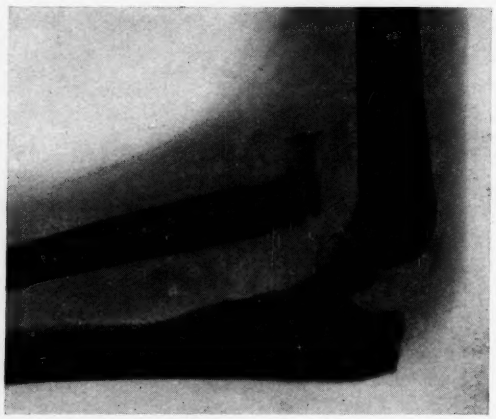


FIG. 32.—*Case* 6. Congenital dislocation of the head of the radius. Note the ossification in the capsule. (Cf. *Figs.* 33, 34.)

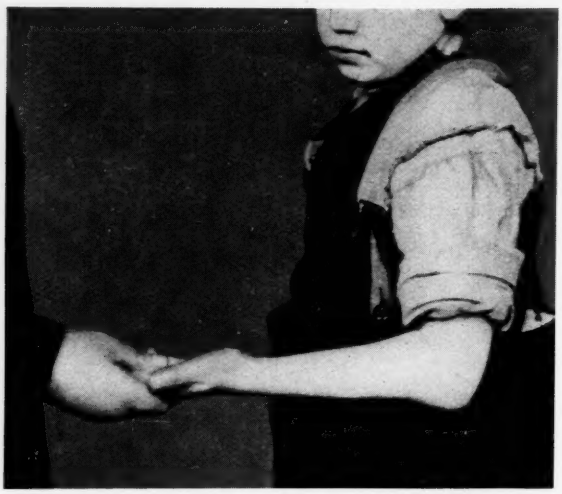


FIG. 33.—*Case* 6. Photograph of congenital dislocation of the head of the radius. Note the anterior curve of the posterior outline of the ulna. (Cf. *Fig.* 34. *See Fig.* 32 for radiograph.)

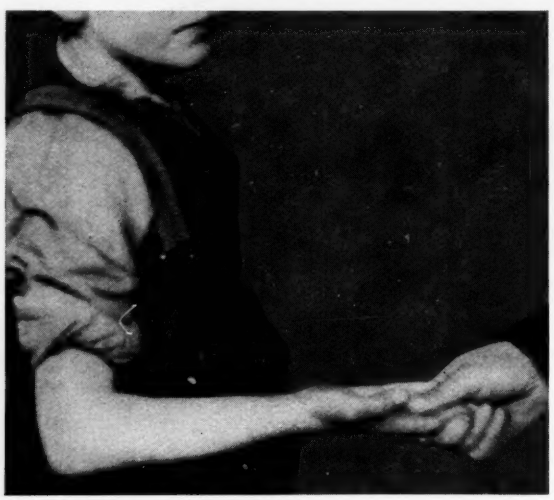


FIG. 34.—*Case* 6. Photograph of normal elbow. (Cf. *Fig.* 33.)

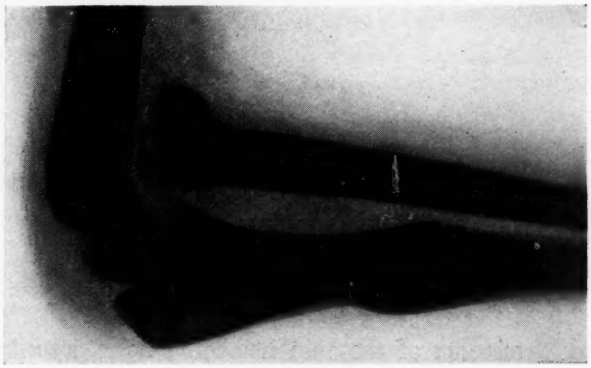
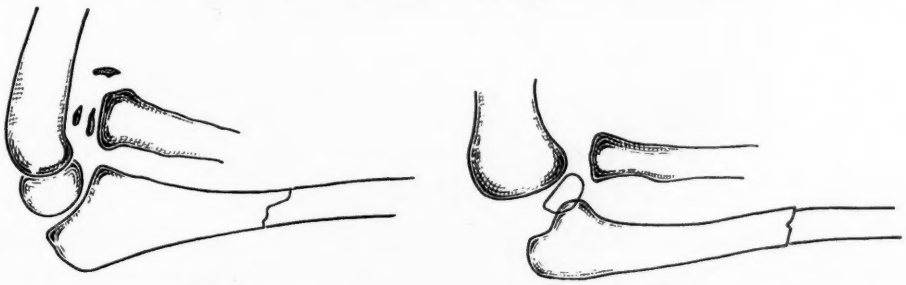


FIG. 35.—*Case* 7. Fractured ulna and congenital dislocation of the head of the radius. This diagnosis may seem unjustified, but *Fig.* 36 shows an elbow of a child nearly the same age in which the dislocation was traumatic and associated with fracture of the ulna. That the changes typical of congenital dislocation are present in the ulna of *Fig.* 35 and not in *Fig.* 36 is made more clear in *Fig.* 37.

FIG. 36.—*Case* 8. Traumatic dislocation associated with fracture of ulna. Age only slightly less than *Case* 7. (Cf. *Fig.* 37.)



Case 7. *Case* 8.

FIG. 37.—*Cases* 7 and 8 contrasted. The deformity of the ulna due to fracture has been discounted by tracing the radiograph so as to reconstruct the ulna.

SUMMARY

A congenital dislocation of the head of the radius is described, setting out the clinical, radiological, and operative observations on eleven cases. The dislocation was in each instance anterior and unilateral. In only five patients was function interfered with and operation undertaken. Removal of the head is preferable to replacement except under certain specified conditions.

MULTIPLE PRIMARY EPITHELIOMA IN LYMPHATIC LEUKÆMIA

By R. J. V. PULVERTAFT

DIRECTOR OF PATHOLOGY, JOHN BURFORD CARLILL PATHOLOGICAL LABORATORIES, WESTMINSTER HOSPITAL

MULTIPLE primary carcinoma of the skin has often been described. It occurs in X-ray workers and in xeroderma pigmentosum, and is particularly associated with those whose work, as in the case of sailors, leads to constant exposure to the elements. In the following case the occupation of the patient, a grave-digger, brings him into this category. The case is, however, unusual in that his cutaneous condition was in part carcinomatous, and in part a complication of lymphatic leukæmia.

The patient, a grave-digger, aged 66, was admitted to Westminster Hospital in March, 1929, with a three months' history of ulcer of the lower lip. A portion

FIG. 38.—Section of tumour of lip, March, 1929. Squamous-cell carcinoma.

was removed, and showed a typical well-keratinized squamous-cell carcinoma (*Fig.* 38). The corium was densely infiltrated with lymphocytes. There were a few submental glands. He received 936·8 mg.-hr. radium to the lesion, and 1685·6 mg.-hr. to glands. In August, 1929, the original lesion was apparently cured; there were some palpable cervical glands, and these received 3007·6 mg.-hr. of radium. They returned practically to normal size.

No blood examination was performed at the patient's first admission, and the tumour of the lip was diagnosed as a squamous-cell carcinoma without qualification. Later, however, it was re-examined in the light of the multiple lesions, and the histology appears identical with them. The amount of lymphocytic infiltration of the corium was very much greater than is normally found in squamous-cell carcinoma, although, of course, such infiltration is almost always present to some extent.

On admission in August, 1933, the patient stated that he had remained well until April of that year, when a small nodule appeared in the skin of the right malar region. It became larger and broke down in two months; it never healed. Following this lesion many others of similar nature appeared elsewhere, on hands

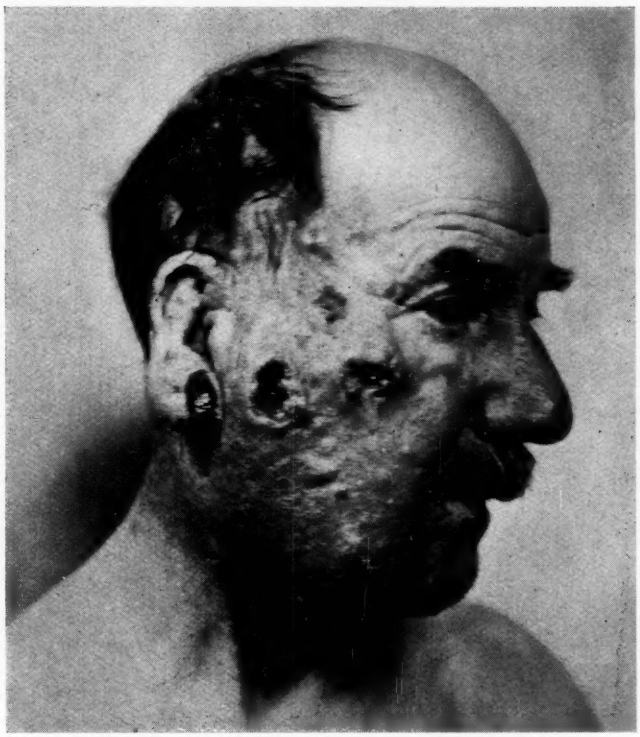


FIG. 39.—Appearance of face, August, 1933.

and face, and followed the same course (*Figs.* 39, 40). Œdema of the face followed the appearance of the malar lesion. He complained of nothing else.

He was seen by Dr. S. E. Dore, who reported, " Lesions on face consistent with those sometimes found in lymphatic leukæmia, but the ulcers resemble epitheliomata. The condition of the skin on the back of the hands is like that of ' sailor skin ' produced by exposure. The recent tumour formation (on the hands) resembles that of the face."

The original lesion of the lower lip now showed soft and painless skin of scar-tissue type. On the right side of the face were numerous ulcers with raised and thickened edges, and depressed central pits showing no attempt at granulation. There was marked œdema of the right side of the face; the skin was pendulous

over the mandibular area. There were many enlarged lymph glands in the right cervical chain; the glands on the left side, previously treated by radium, had not increased in size. The superficial lymph glands all over the body were readily palpable. There was a large scrotal hernia. Neither liver nor spleen was enlarged. The hands and arms showed lesions similar to those on the face. The rest of the skin was normal; there were no purpuric lesions and the patient did not complain of pruritus. There were numerous retinal hæmorrhages. There were no other abnormal physical signs. Blood examination showed: R.B.C. 1,910,000, Hb 23 per cent, C.I. 0·6, W.B.C., 226,400. All the cells seen were primitive lymphocytes, with prominent nucleoli. The Wassermann reaction was negative, and the urine normal.

FIG. 40.—Appearance of arms and hands, August, 1933.

A biopsy was performed by Mr. E. Rock Carling, and showed the following results. (*Figs.* 41, 42.)

Axillary lymph gland. Much enlarged. The germinal centres are not visible, and the general structure altered. It consists exclusively of small deeply-staining cells of the general appearance of lymphocytes. There is no evidence of squamous-cell carcinoma, and the picture is typically that of lymphatic leukæmia.

Small non-ulcerated lesion of back of hand. Pronounced œdema of cutis vera. The epithelium is much thinned, the papillæ are much prolonged, and there is diffuse lymphocytic infiltration. The whole picture is that of psoriasis.

Two lesions of hand, and large ulcer of face, central area. Typical well-keratinized squamous-cell carcinoma, penetrating deeply. There is a massive infiltration with lymphocytes, but this is frequently found in normal squamous-cell carcinoma.

Large ulcer of face, periphery. The epithelium is very thin, and there is a dense infiltration with lymphocytes extending throughout the corium and infiltrating

the fat. The papillæ are absent as in scar tissue. There is also an area of early squamous-cell carcinoma.

The patient left the hospital and died at home; there was no autopsy.

FIG. 41.—Section of tumour of face, August, 1933. Leukæmic deposit in skin with early squamous-cell carcinoma.

FIG. 42.—Section of lesion of hand, August, 1933. Thinning of epithelium; downward processes elongated.

DISCUSSION

Lymphatic leukæmia is usually considered to be a neoplastic disorder, and the association of two distinct malignant conditions, while of interest, is familiar enough. The chief point of note in this case is its bearing on the function of the lymphocyte in malignancy. Murphy has published much work on this subject, starting from the observation that there is consistently a lymphocytic infiltration at the growing edge of a malignant neoplasm. He argued that the lymphocyte provided an important barrier to the spread of tumours.

There is, however, evidence leading in the opposite direction. In cutaneous epithelioma invasion only occurs into tissues where lymphocytes are aggregated; and lymphatic glands are of all organs the most prone to secondary invasion.

Leukæmic deposits in the skin are of frequent occurrence, and take many forms. The condition is fully described by Hirschfeld. In a long series of references no allusion is made to the development of malignant changes in the superficial epithelium, and it has not been possible to find any case of such a change. It seems probable, therefore, that the primary exciting factor for the epitheliomata was the long-standing condition of xeroderma. In every case, however, the epitheliomata in fact developed in areas of leukæmic infiltration, and this suggests that nutritional or other changes occasioned by their presence were, for the already pathological epithelium, the last straw. It is certainly remarkable that epitheliomata consistently develop in areas affected by injuries of long standing, where subepithelial fibrosis is a feature; we find this in lupus, old burns and scars, and syphilis. There may be a purely nutritional basis for this fact; and the leukæmic infiltration may have precipitated in this case malignancy in which a defective blood-supply to the epithelium was one factor.

My thanks are due to Mr. E. Rock Carling for permission to publish this case, and to Dr. R. G. Waller for the photographs.

REFERENCES

HADFIELD, G., and GARROD, L. P., *Recent Advances in Pathology*, 1932, 84. London.
HIRSCHFELD, H., *Krankheiten des Blutes*, 1925, i, Berlin.

ACUTE ILIO-PSOAS ABSCESS

By R. E. NORRISH
SURGICAL REGISTRAR, ROYAL NORTHERN HOSPITAL, LONDON

In recent years suppurative conditions within the sheath of the ilio-psoas muscle have usually been assumed to be tuberculous and derived from some part of the vertebral column or from an associated joint. There is, however, a subacute or acute lesion, characterized by the formation of a large abscess, from which pyogenic organisms may be readily obtained. The origin of such an abscess may be from any one of a variety of sources; it may be a blood- or lymph-borne infection, or may be the result of a direct extension through the sheath from a neighbouring associated structure. The case described below belongs to the last group, being the result of a perforation of the cæcum, and in one or two of its features would seem to be unique.

History.—The patient was a man of 62 years, who for three months had complained of vague central abdominal pain. There had been general malaise and some loss of weight, with marked flatulence. He had not noticed nausea or sickness, and the bowel had been opened twice or three times daily during several previous weeks, the stool being loose, but otherwise normal. During this period he attended as an out-patient, and a radiological examination of the gastro-intestinal tract was carried out. No abnormality was detected.

Two or three days before his admission to hospital, the pain had become more severe, all over the right side of his abdomen, from the costal margin to the groin, being most severe in the lower quadrant. He had not vomited, though diarrhœa had supervened; micturition was normal.

On Examination.—The patient's temperature was 99·8° and his pulse 88, with respirations 23 per minute. He was a thin, ill-nourished man, with muddy complexion and moist, slightly furred tongue. The abdomen, poorly covered, showed diminished respiratory movement and tenderness over the whole of the right side, increased below the umbilicus and reaching a maximum in the groin. The tonus of the abdominal musculature was increased a little in the right lower quadrant, though true rigidity was absent. There was no palpable swelling. Rectal examination showed no abnormality, and the urine was clear and normal. The right thigh was held in slight flexion and external rotation at the hip-joint, the contour being normal.

The patient was placed under observation, and during the succeeding twenty-four hours his pulse and temperature remained steady. The pain in his abdomen diminished in the upper part, but increased in the groin, the increased tonus persisting, but in lesser degree. The thigh became more flexed and rotated outwards and some swelling became evident. This was at first seen as a fullness in Scarpa's triangle, spreading gradually to the inner and posterior aspects of the limb. Attempts to extend the thigh and rotate it inwards were strongly resisted by the patient, though further flexion was not resented.

Forty-eight hours after his admission, the abdominal signs had largely disappeared, and the focus of his complaint seemed to have moved to his thigh. There was still no nausea or sickness, though the diarrhœa persisted, the stools being free from blood. The thigh had become increasingly swollen, and œdema appeared over Scarpa's triangle and over the adductor area. The limb lay heavily on its outer side, now flexed to fully a right angle at the hip. Percussion upon the greater trochanter failed to elicit pain, and there was no clinical evidence of lesion in either the spine or bony pelvis. This was confirmed by radiological examination. An X-ray photograph of the right hip-joint, however, showed a collection of gas around the neck of the right femur (*Fig.* 43), and it was concluded that an appendix abscess

FIG. 43.—X-ray photograph of the right hip-joint, showing a collection of gas around the lesser trochanter and neck of the femur. The abduction and external rotation at the joint are evident.

was presenting in the thigh, having passed along the psoas sheath. Neither swelling nor fullness was palpable above the inguinal ligament.

OPERATION.—The general condition of the patient was very poor; the pulse was of low tension and had risen a little, and it was felt that he would successfully withstand only the minimum of surgical interference. Accordingly, under gas and oxygen anæsthesia, an aspirating needle was passed into the swelling in Scarpa's triangle, to the outer side of the femoral vessels. At once gas with fæcal odour escaped, and thin sanious pus was aspirated. With the needle still in position, a free vertical incision was made into the abscess cavity; this was extensive, passing

beneath the femoral vessels, down to the neck of the femur and between adjacent muscles. The wound was left widely open, being lightly packed with gauze. Bacteriological examination of the pus showed coliform bacilli and streptococci, and a profuse growth of these organisms was obtained.

During the next few days the general state of the patient deteriorated further. A profuse discharge occurred from the wound, purulent at first, fæcal later. He died on the tenth day after his operation.

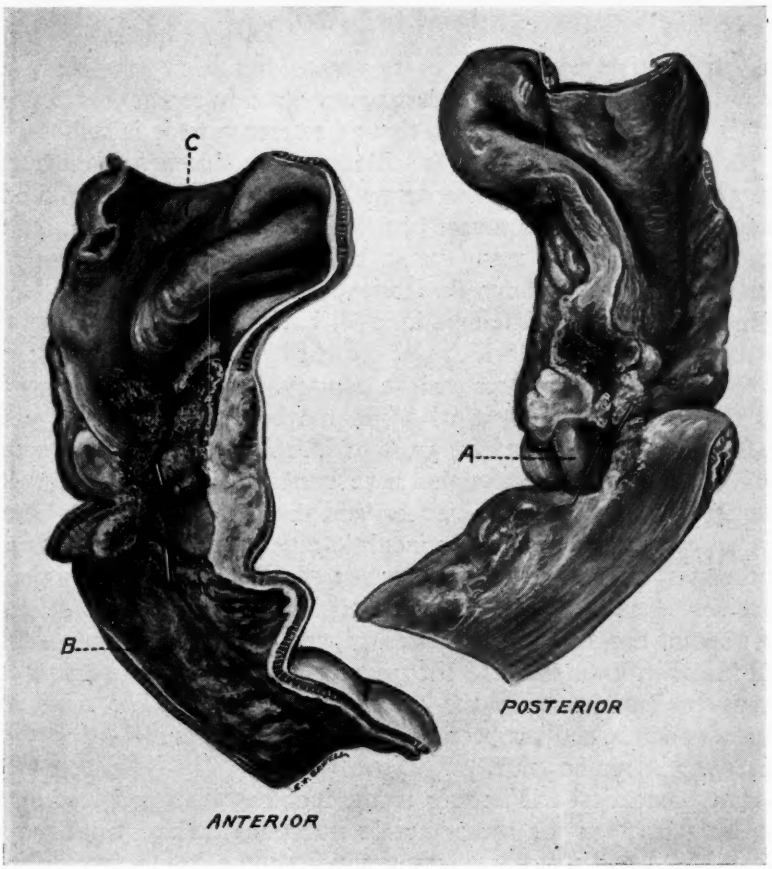


FIG. 44.—Carcinoma of cæcum, enveloping the appendix, and showing two perforations. A, Appendix; B, Ileum; C, Ascending colon. (× ⅜.)

AUTOPSY.—A small carcinoma was found in the cæcum (*Fig.* 44). It was placed upon the posterior wall of the bowel and infiltrated the lateral walls, projecting a little above the surface of the mucosa. Its base showed ulceration and a small double perforation. The appendix was almost completely enveloped in the neoplasm, its tip being the only recognizable part. A large abscess cavity was situated behind the cæcum, extending outwards for a little way across the iliacus, and downwards to the thigh, within the psoas sheath. The psoas was represented by a dark confluent mass, with pus around and within it, and led downwards beneath Poupart's ligament to a large excavated space in the upper part of the thigh. This

cavity extended on each side of and beneath the femoral vessels, and led down to the medial side of the femoral neck. The vessels and nerves stood across it, cleanly dissected, and the space could be traced among the muscles around it. It contained a brownish, odorous pus.

Microscopic section of the tumour showed a columnar-celled carcinoma, with alveolar formation and with abundant fibrosis. Its origin, whether from the cæcum or proximal part of the appendix, is uncertain.

DISCUSSION

Acute suppuration within the psoas sheath has been very fully described, especially during the latter half of the last century, and in recent years a considerable number of cases have been recorded, though reference to it is uniformly omitted from our surgical text-books. In 1878 Margano[1] produced a monograph dealing with acute psoas infection following trauma. Many examples are cited therein, with abundant evidence of the source of the abscess. The author traces references to records which appeared many years before his time. Apparently the first description was made by Fabrice de Hilden,[2] who, in a letter in 1682, tells of a young man who tripped and fell backwards, bruising his back. There followed an abscess in his psoas muscle which was opened by his surgeon. In 1797 Déramé described fully a similar case, proved at autopsy.

Acute psoas abscess of intestinal origin has been recognized for many years, and among the earlier references are those of Ménière in 1828, and Dance in 1832. Since that time many more examples have been recorded, but they all refer to typhlitis or appendicitis as the forerunner, and thus occurred on the right side.

Direct spread of pus from a perinephric abscess, from parametritis, from an empyema thoracis, or from acute osteomyelitis of the ilium, has led to the formation of a massive abscess in the thigh. A psoitis arising in the course of a general pyæmia has in more recent times been fully described, and cutaneous suppuration and naso-pharyngeal infection have supplied the primary focus, with the staphylococcus and streptococcus predominating as the causative organisms. The condition is not uncommon in children, and has been described by Ingelrans and Minne,[3] Behrman,[4] and Sworn,[5] within recent years.

The close anatomical relationship to the inguinal, iliac, and lumbar lymphatic glands has brought upon the psoas muscle infections derived from the lymphatic areas drained by those systems, particularly from the lower limb. The lumbar retroperitoneal lymph nodes have been especially blamed by Rogers,[6] though his evidence is not very convincing.

Pus may be localized to various parts of the psoas sheath, and occasionally it spreads out across the iliacus muscle. Typically, the fully developed abscess presents a deep and tender swelling above the inguinal ligament connected to a second below that structure. Passing out of the sheath at the neck of the femur, an extensive suppuration often burrows among the flexor and adductor muscles of the thigh, and so may pass on down the limb. On opening such an abscess below the inguinal ligament, there is displayed a cavity which may be wide-spread among the muscles, across which the femoral vessels may stretch unsupported, and in which the femoral nerve, dividing into its many branches, may stand out cleanly dissected. As in other forms of psoas abscess, pus may

reach to a point as distant as the heel before presenting. An interesting record is made by Layton,[7] who described an example in which, on account of long recumbency, the reverse process occurred. A popliteal adenitis derived from erysipelas of the heel resulted in an abscess which tracked upwards through the thigh to Scarpa's triangle, there entered the psoas sheath, and ascended through the abdomen to the thorax.

The flexed and externally rotated thigh is characteristic and increases as the condition advances. It has been said that " where the massive thigh lies flexed and immovable upon its outer side, a fatal termination is to be expected, however free the drainage that be instituted."

I am indebted to Mr. F. D. Saner for kindly giving me permission to publish this case.

REFERENCES

[1] MARGANO, G., " La Psoïte traumatique ", *Trans. Roy. Med. and Chir. Soc. Paris*, 1878.
[2] FABRICIUS HILDANUS, *Observationum et Epistolarum Chirurgico-medicarum*, 1716, i.
[3] INGELRANS, P., and MINNE, J., " La Psoïtis de l'Enfant et de l'Adolescent ", *Arch. franco-belges de Chir.*, 1930, xxii, Dec., 1930.
[4] BEHRMAN, S., " Non-spinous Ilio-psoas Abscess ", *Lancet*, 1930, ii, 297.
[5] SWORN, B. R., " Acute Psoas Abscess ", *Brit. Med. Jour.*, 1933, ii, 6.
[6] ROGERS, M. H., " Psoas Abscess from Lumbar Retro-peritoneal Lymph Glands ", *Amer. Jour. Orthop. Surg.*, 1911, ix, 232.
[7] LAYTON, T. B., " An Unusual Case of Psoas Abscess ", *Guy's Hosp. Gaz.*, 1912, xxvi.

CLEFT STERNUM: CASE REPORT AND BRIEF COMMENTARY

By GLEN A. M. KNIGHT and GEORGE H. MORLEY
ROYAL AIR FORCE HOSPITAL, ADEN

COMPLETE median cleft or fissure of the sternum has been described by various observers, usually associated with defects of the neck and abdominal wall. The authors feel that the case to be described here, having no detectable abnormality other than the sternal deficiency and a superficial scar of the abdominal wall, is therefore worthy of record.

HISTORICAL SURVEY

Gibson and Malet[1] (1879) described a case in which a sternal fissure existed in the cranial end of the bone as far downwards as the upper border of the fourth rib; this was associated with a strong membrane attaching the sternal edge in the cleft to the cricoid cartilage, and a scar-like strip of skin, 3 in. long, passing upwards from the umbilicus. Professor Sir William Turner[2] (1879) described a specimen in the Anatomical Museum of the University of Edinburgh, with fissure of the sternum ending at the level of the seventh rib, where the two halves articulated in the mid-line. He quoted cases of Herr Groux, Skoda and Forster, the former describing, apparently, his own condition, and the latter found the ribs to be fused to the sternum without articulation.

Martin[3] (1887) described a case in a new-born female, which he traced to be healthy to the age of $4\frac{1}{2}$ months, though with umbilical hernia, divarication of the rectus muscles, and attenuation of the skin of the precordial area.

More recently Barillet[4] described a case in which the cleft was sufficiently large to permit examination and direct record of the aortic pulse. Benhamou, Hermann, and Levi-Valensi[5] have described a case of apparent complete absence, as opposed to fissure, of the sternum, with records of cardiograms obtained directly through skin from the pericardial surface of the heart as far out as the apex. It is interesting to note that no cardiac abnormality could be detected.

The condition was exhaustively reviewed in 1922 by Alfred Szenes[6] and in 1926 by Mr. David Greig, of Edinburgh.[7] Szene's case was a male aged 8 years, with complete fissure of the manubrium and body of the sternum as far as the xiphoid at the sixth costal cartilages. Scarred skin lined the fissure and a ligamentous raphe connected a spur-like protuberance of the chin with the sternal fissure, over which it spread fan-wise. This raphe caused a pouch-like fullness of the neck and limited extension of the head on the neck.

Morton and Jordan[9] (1935) described a still further stage in a case of median cleft of the lower lip and mandible, cleft sternum, and absence of the basi-hyoid. In their case the sternal deformity consisted of apparent absence of the manubrium and upper half of the sternum, with the inferior gnathoschisis and the connecting area of scar tissue from the mental region to the floor of the sternal cleft.

CASE REPORT

A male Arab, aged about 20 years, weaver, of Shaikh Othmann, Aden, was seen by Dr. Napier, of the Keith Falconer Hospital, in November, 1935, on account of ulcers of his legs. In the course of examination Dr. Napier discovered the condition to be described, and very kindly brought it to our notice.

The condition—apparent absence of the sternum—had been present since birth; there were no relative symptoms. The patient and his parents were, in fact, surprised that it should evoke more than passing interest. Family history, as far as could be ascertained, revealed no other case with congenital abnormality of any kind.

On Examination.—The patient was somewhat below the normal stature for his age, and of a poor type mentally; the general development was otherwise a fair average of the local type of Arab and he followed his employment regularly. Ulcers were present on the lower halves of both legs, not associated with varicosities or œdema of the feet, and appeared to be of the tropical type which is common amongst the natives of this district.

On the anterior aspect of the thorax in the mid-line, the area normally occupied by the sternum appeared to be replaced by a well-marked vertical cleft (*Figs.* 45, 46) which is described

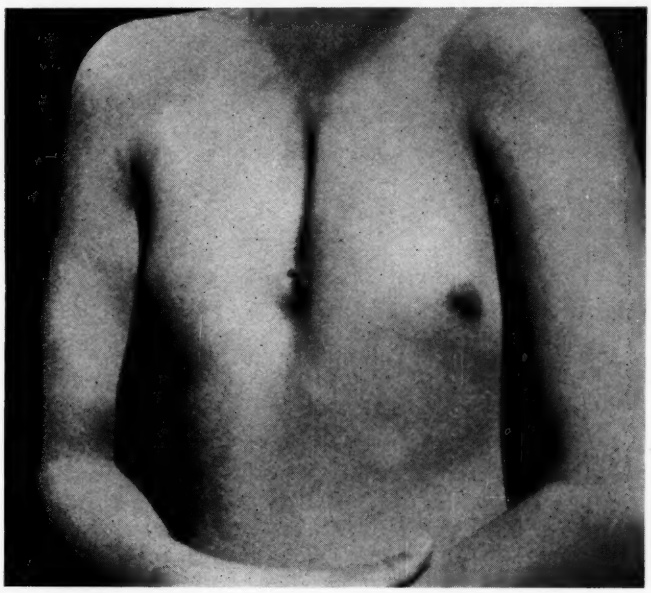


Fig. 45.—Photograph showing condition of sternum.

in detail below. There was, in the floor of this depressed area, opposite the fourth costal cartilages, a circular, puckered scar, ½ in. in diameter, soundly healed and not adherent to deep structures.

In the mid-line of the abdomen was a slightly raised linear scar, 2 in. long, ending below at the umbilicus. Both scars had been present since birth. There was no history of trauma. The recti abdominis muscles were normal, there was no herniation at the umbilicus, and the symphysis pubis was normal.

The mouth, tongue, and mandible were normal, as was also the neck. A degree of deficiency of pubo-axillary hair was noted, but otherwise the pudenda were normal. No other abnormality could be detected on examination.

The Sternum.—The manubrium and body of the sternum were found to be deficient in the mid-line as far, inferiorly, as the xiphisternum; laterally, remains of these portions were present, so that the manubrium and body of the sternum were, in effect, split into two portions by sagittal section. Integument passed into the cleft so formed, and, except for the puckered scar noted as being opposite the fourth costal cartilages, was of normal texture. A regular pulsation was visible in the lower third of the cleft.

Mensuration.—In the resting decubitus position the space between the inner sternal edges at the upper end of the cleft was 1½ in.; during full inspiration this increased to 1¾ in. and it decreased to 1¼ in. during full expiration. In the sitting position, at rest, this measurement was 1 in. At the nipple line, in resting decubitus, the space was 1½ in. wide, whilst at the lower end, opposite the sixth costal cartilages, the space measured 1 in.; the latter measurement did not vary with position or with respiration.

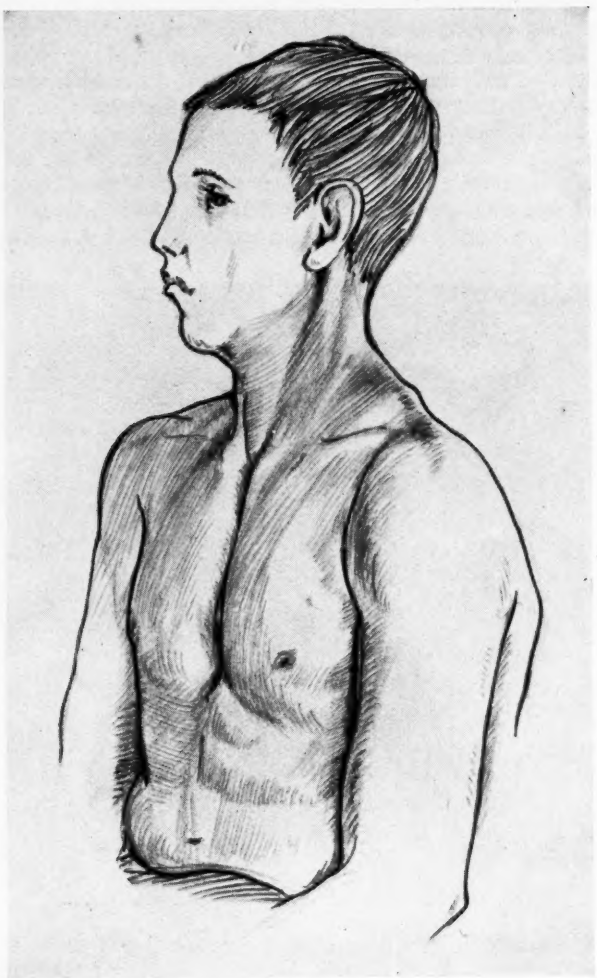


FIG. 46.—Another view of the condition. *(From a pencil sketch by Mrs. Haythornthwaite.)*

Anatomy.—

The borders of the cleft.—

Superior: No border could be defined and the cleft passed without interruption into an apparently normal neck.

Inferior: A definite, bony-hard margin, concave superiorly, at the level of the sixth costal catilages; this appeared to be firmly attached to the lateral borders of the cleft, and on palpation resembled the upper border of a small ill-defined xiphisternum, the total length of which was not more than ½ in.

Lateral: Smooth osseous edges, ¼ in. thick, extended on both sides the full length of the cleft; to the lateral margins of these osseous plates, which were about ½ in. wide, were attached, in normal disposition, the clavicles and costal cartilages.

The floor of the cleft.—The depth of the floor, below the level of the adjacent chest wall, was seen to vary with respiration: in the resting position one finger could be laid in the cleft and was then flush with the edges. In expiration a degree of shallowing occurred, whilst during the act of coughing the floor rose above the level of the surrounding edges so that considerable herniation of the pleura or mediastinum occurred.

The deep relations of the floor, from above downwards, were: Trachea, deeply placed and covered by soft connective tissue. Just below the level of the second costal cartilages a definite fibrous band could be felt crossing transversely; this appeared to be the upper border of a thickening of fibrous tissue some 1 in. wide from above downwards, the lower border being less definite and apparently merging into the pericardium. From this lower border to the upper edge of the xiphisternum the floor consisted of pericardium covering heart and great vessels, the determination of the exact anatomy of these being indefinite. The pulsations were, however, obvious on first looking at the patient, and, on palpation, the contractions could be appreciated just under the fingers: deeper palpation in this area was not tolerated by the patient.

THE CHEST.—Circumference at the nipple line, resting, was 33 in. The full respiratory excursion was only $\frac{3}{4}$ in. Clinical examination revealed no abnormality in the pulmonary or in the cardiovascular system. These findings were confirmed by X rays, the latter showing also the outline of the cleft sternum until in the lower half it is obscured by the cardiac shadow.

ALIMENTARY SYSTEM.—Mouth normal; the position and function of the œsophagus, stomach, and duodenum were examined by a small barium meal and observed by screening to be normal.

The Meinicke reaction was strongly positive; no other abnormality could be discovered in the examination of the patient.

COMMENTARY

Development.—The sternum develops, according to Keith,[8] as a condensation of mesoderm on each side of the mid-ventral line, then represented by the primitive linea alba, at the fifth week. These form the right and left mesenchymal sternal bars, which are continuous anteriorly with the bases of the ventral part of the shoulder girdle, and are situated over the pericardium between the mandible in front and the umbilicus behind. Fusion of these sternal bars in the mid-line commences at the presternum about the 13-mm. stage (end of sixth week); the caudal portions are kept apart by the protrusion of the heart and liver until union takes place by the 34-mm. stage (tenth week). Chondrification and ossification then proceed.

Defects.—Bifid xiphisternum is common, whilst occasionally a median foramen is found in the region of the lower sternal segments. Fissure of the manubrium, extending a variable distance into the body, would appear to be a more usual occurrence than complete fissure of the sternum. Deficiency of the caudal end only is found in association with a gross embryonic lesion such, for example, as ectopia cordis.

Causative Theories.—Szenes,[6] reviewing the point, favours some cause of obstruction operating between the cranial ends of the sternal bars, about the 15-mm. stage, relieved at a later stage to allow union of the caudal portions. He puts forward, as this cause, pressure, probably due to deficiency of amniotic fluid, and adhesion between the chin and chest wall during the stage of acute flexion, relieved later by extension of the head. In support he quotes a case of von Mall of an anencephalic embryo with the face adherent to the chest wall. Morton and Jordan[8] appear to have arrived at a similar conclusion, and stress the importance of transient adhesions rather than pressure.

The case described in this paper would appear to be one of complete sternal fissure with fewer complicating features than we have been enabled to trace in other cases published. The scarring described may be regarded as a result of those influences which in other cases have produced gross lesions, and in this case failed to do so. The influence of some external or ectodermal (amniotic) factor seems to be supported, and the differentiation of these cases from defects of inclusion of viscera, such as ectopia cordis, made clear by the fact that the defect is usually in the cranial end of the sternum, and not in the caudal end as might be expected in these cases on embryological grounds. It is the caudal end which Keith[9] and others maintain is last to unite, on account of the protrusion of the large heart and liver.

The time at which this defect originates may be supported as that at which the chin, chest wall, and umbilicus are closely apposed, synchronous with union of the cranial ends of the sternal bars, and separated in time to permit the caudal ends to come together, i.e., between the 13-mm. and 30-mm. stages.

Clinically, interest lies in the fact that no symptoms arose from this condition and that interference was not indicated for the complete sternal defect *per se*. The heart was not embarrassed, and we suggest that the thickened fibrous fold described in the floor of the cleft is a strengthened superior sterno-pericardial suspensory ligament which is described as arising normally from the dorsal surface of the angle of Ludwig.

In conclusion we wish to thank Dr. Napier for having shown us this case and inviting us to carry out investigations; and to express our sincere gratitude to Professor Grey Turner, who, in a short visit to Aden, found time to examine the case and to aid and encourage us by his valuable opinion and helpful advice in the preparation of this paper. We would also thank the Officer Commanding, Royal Air Force Hospital, Aden, for providing us with facilities for the examination and investigation of this case.

REFERENCES

[1] GIBSON and MALET, *Jour. Anat. and Physiol.*, 1879–80, xiv, 1.
[2] TURNER, *Ibid.*, 103.
[3] MARTIN, *Med. Record*, 1887, xxxii, 425.
[4] BARILLET, *Comptes rend. Soc. de Biol.*, 1929, ci, 829; *Presse méd.*, 1929, No. 50, 817.
[5] BENHAMOU, HERMANN, and LEVI-VALENSI, *Comptes rend. Soc. de Biol.*, 1929, cvii, 168.
[6] SZENES, *Arch. f. klin. Chir.*, 1922, cxix, 116.
[7] GREIG, *Edin. Med. Jour.*, 1926, Aug., 480.
[8] MORTON and JORDAN, *Arch. of Surg.*, 1935, xxx, 647.
[9] KEITH, Sir A., *Human Embryology and Morphology*, 5th ed., 1933, 472. London.

MELANOMA OF THE RECTUM*

By HAROLD H. LINDNER, San Francisco
and W. QUARRY WOOD, Edinburgh

Melanoma of the rectum is an uncommon disease, but probably less so than would appear from an examination of the literature. It is a condition of peculiar interest, particularly from the point of view of its pathogenesis and its relationship to the other forms of melanomata. Melanomata of the skin and the eye are well known, and rare cases arising from derivatives of the neural ectoderm, especially in the central nervous system and the suprarenal medulla, have been described. In the alimentary canal, apart from the rectum, melanomata are exceedingly rare. In his admirable and most exhaustive monograph on the melanomata, Dawson makes no specific mention of the occurrence of this tumour in the rectum, and we have not been able to find any detailed account of the disease in English literature, although a certain number of cases have been briefly recorded. We therefore feel that a recent case which we were able to study in detail should be put on record.

HISTORY

Melanomata of the rectum were recognized in the lower animals for many years before they were known in man. They were described in the horse before the end of the eighteenth century. Laennec, in 1806, isolated and described the ordinary forms of melanomata in man, but it was not until 1857 that a case of melanoma of the rectum was recorded by Moore. Virchow, in 1869, mentions a case reported by Maier, and adds that he had seen one case himself which ran a very malignant course. The first complete record of a case was that of Paneth in 1883; he described fully a personal case and mentioned 8 others recorded up to that date. From that time onwards records became more numerous, and in 1913 Chalier and Bonnet, in a very excellent and comprehensive review of the whole subject, were able to collect and analyse the records of 64 cases, including one of their own; they mention 8 other possible cases of which they had heard but which they had been unable to trace. This paper by Chalier and Bonnet is much the most satisfactory, and indeed is the only complete, article on the subject in modern literature. In 1918 Churchman in reporting a case had been able to collect only 2 more since 1913, bringing the number of authenticated cases up to 67. Since 1918 we have been able to find only 11 cases in the literature, those of Kraker, McGuire and Leahy, Goldblatt, Allan, Kallet and Saltzstein (3 cases), Landman, Gerritzen, Marino, and Ingle. This brings the total of recorded cases up to 78, with 8 others mentioned but untraced by Chalier and Bonnet.

---

* From the Department of Surgery, University of Edinburgh.

CASE REPORT

The following case was operated on by one of us as a malignant tumour of the rectum, but its true nature was not recognized until the routine examination of the operation specimen was carried out. The patient was a married woman, aged 53. She had complained of pain in the rectum and passage of blood on defæcation for a period of four months. Constipation was not a prominent feature, and her general health had remained satisfactory. On digital examination of the rectum an indurated swelling was encountered; it was situated at the level of the ano-rectal junction and was mainly on the posterior wall. It projected into the lumen of the bowel but did not prevent the passage of the finger. There was no enlargement of the inguinal glands, and abdominal examination did not reveal any abnormality. The condition was regarded as an adenocarcinoma of the rectum of the ordinary type.

OPERATION.—At the end of August, 1935, an inguinal colostomy was performed. The liver was examined and found to be smooth on the surface and free from any palpable evidence of secondary growths. There was no evidence of metastases in the peritoneum and no enlarged glands were found in the pelvic mesocolon or in relation to the rectum. The tumour was not adherent to the perirectal tissues and was considered to be eminently suitable for a radical operation.

On Sept. 8, 1935, under general anæsthesia, an excision of the rectum was carried out by the perineal route. The coccyx was excised, the peritoneal cavity opened freely, and the bowel divided through the lower part of the pelvic colon. The peritoneum and the proximal end of the colon having been closed, the cavity was packed with gauze and allowed to heal by granulation. The patient made a good recovery from the operation. She was examir in April, 1936, and was found to be in good health. No gross evidence of metastasis coula be found.

MORBID ANATOMY AND HISTOPATHOLOGY

The true nature of the tumour was only recognized in the course of the routine examination of the operation specimen. The method employed has been previously reported. The site and size of the growth and the condition of the lymphatic glands as regards infection by the growth are recorded in the chart shown in *Fig.* 47. After dissection of the lymphatic glands a section was made through the specimen in the sagittal plane. This showed a large blackish-brown tumour arising from the upper part of the anal canal and the lower part of the rectum proper. The appearance of the cut surface of the tumour with its anatomical relationships is well shown in *Fig.* 48.

The tumour involved the posterior and lateral aspects of the bowel wall, extending over about two-thirds of its circumference. It showed a very pronounced tendency to bulge into the lumen of the bowel; in the vertical plane the attachment to the bowel wall measured 2·4 cm., while the greatest vertical measurement of the tumour was 4·7 cm. Its base appeared to rest on the muscularis of the lower part of the rectum and the upper part of the anal canal. The tumour was firm and elastic in consistence, but here and there small areas of softening were present. The cut surface of the growth presented an appearance of lobulation, and at one point on the free surface there was an umbilicated depression suggesting the appearance sometimes seen in malignant nodules on the surface of the liver. The free surface of the tumour did not show gross ulceration.

Lying posterior to the main tumour, but separated from it by the muscular wall of the rectum, there was a large rounded nodule of a brownish-black colour. It appeared to be entirely extra-rectal and we were unable to trace any direct continuity between this nodule and the primary tumour. It was enclosed in a well-marked capsule posteriorly, probably derived from the fascial covering or sheath

of the rectum. It measured 4·1 cm. in the vertical plane and 3·4 cm. in the antero-posterior plane. Another much smaller nodule was present about the level of the middle of the base of the main tumour. It also lay outside the muscular coat of the bowel, and measured about 0·5 cm. in the vertical plane. Apart from these two nodules there was no other evidence of spread of the tumour outside the wall of the rectum. There was no pigmentation of the peri-anal skin or of the mucosa of the rectum, apart from some slight brownish discoloration in the immediate neighbourhood of the tumour.

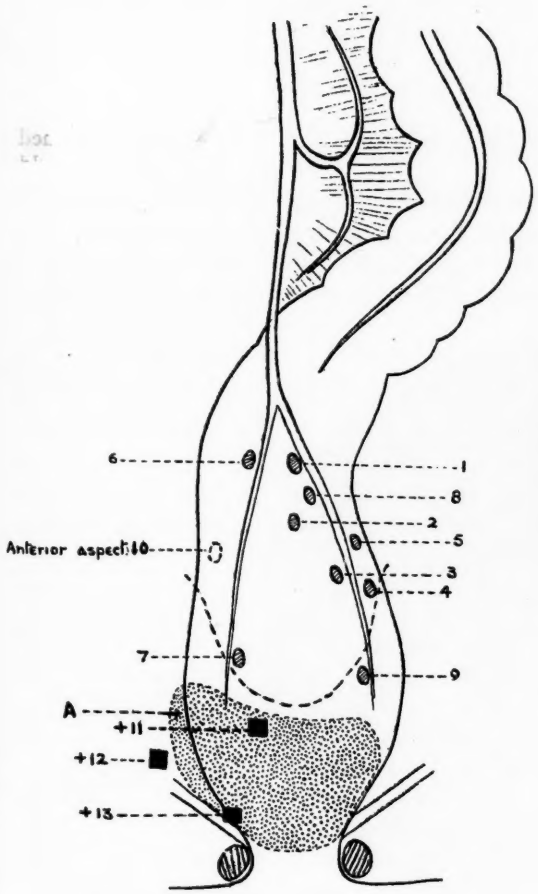


FIG. 47.—Chart showing the position of the tumour and the associated lymphatic glands.

FIG. 48.—Drawing of a sagittal section through the operation specimen. The tumour is growing from the posterior aspect of the bowel.

On dissection of the peri-rectal fat, ten lymphatic glands were found, as indicated in the chart. Nine of these lay on the posterior aspect of the rectum in relation to the branches of the superior hæmorrhoidal artery; the remaining

gland lay on the anterior aspect of the rectum. All the glands were of moderate size, the largest being about 0·5 cm. in diameter, while the majority were about half this size. On external examination, only two of the glands appeared blackish

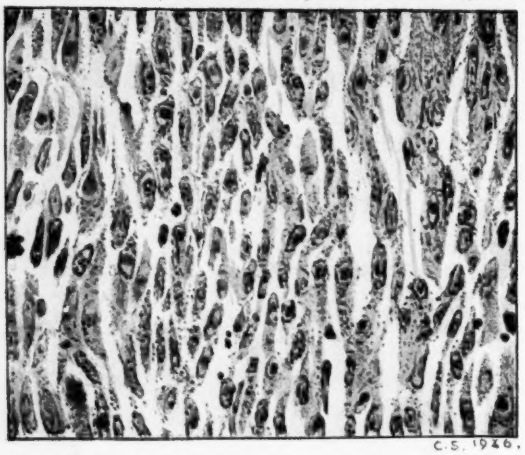


FIG. 49.—Microscopical section of the primary tumour. It is made up largely of spindle cells, presenting a resemblance to a sarcoma.

in colour, but on section all the glands showed some evidence of blackish pigmentation with the exception of the one found on the anterior aspect of the rectum.

Microscopical Appearances.—There was a remarkable difference between the microscopical appearance of the primary tumour and that of the extra-rectal

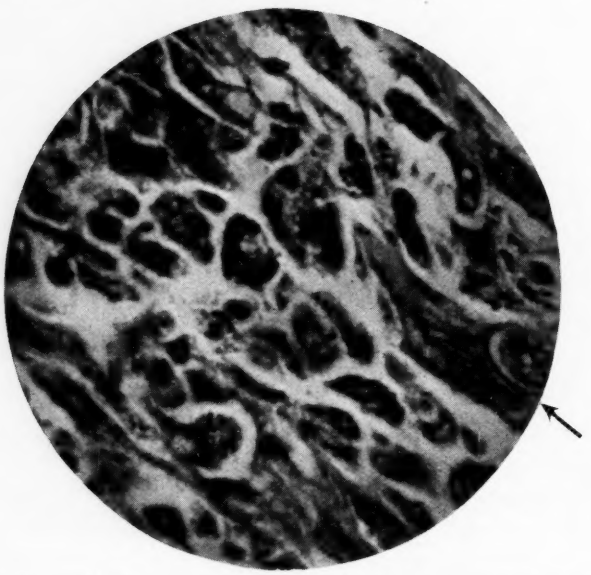


FIG. 50.—Photomicrograph of the tumour showing a large pigment-carrying cell on the right. (× 900.)

nodule. The greater part of the primary tumour was made up of long strands of spindle-shaped cells and presented a close resemblance to the picture of a spindle-celled sarcoma (*Fig.* 49). These elongated cells contained a varying amount of

pigment in the form of fine brownish granules scattered throughout the cytoplasm. Some of the pigment was extra-cellular, suggesting that it had been liberated into the extra-cellular spaces by the breaking down of pigment-containing cells. A considerable proportion of the cells were entirely free from pigment. Although

FIG. 51.—Section through the extra-rectal nodule. The cells are polyhedral in shape with abundant cytoplasm. Pigment granules are numerous.

most of the cells were arranged in strands or columns, in a few areas attempts at the formation of alveoli were seen. A second type of cell was present in small numbers, easily distinguishable from the spindle-shaped tumour cells. It was much larger than the tumour cells, being sometimes two or three times the size, and its cytoplasm was frequently packed with coarse pigment granules (*Fig.* 50). It has

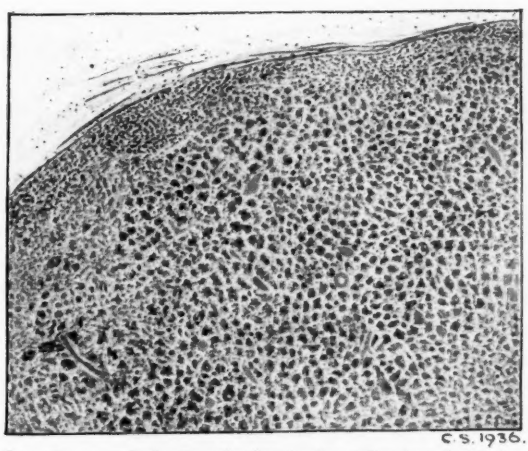


FIG. 52.—Section through one of the lymphatic glands. The lymphoid tissue has been largely replaced by pigment-carrying melanophores.

been shown that this is not a malignant cell but is a phagocytic pigment-carrier. It has been named a *chromatophore* or *melanophore* in contrast to the *melanoblast*, the pigment-producing cell. It was present also in the extra-rectal nodule and in large numbers in the lymphatic glands.

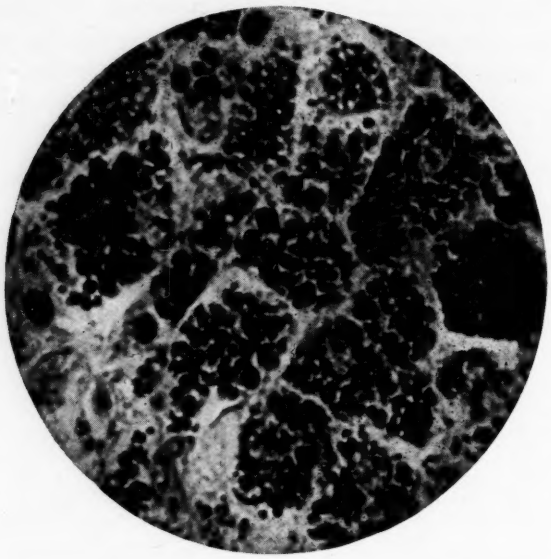


FIG. 53.—A group of melanophores from a lymphatic gland. The cytoplasm is so packed with coarse pigment granules that the nucleus in some of the cells is almost hidden. (× 900.)

FIG. 54.—Large microscopical section through the tumour. This shows the relation of the primary tumour to the rectal wall and the general structure of the primary tumour and the extra-rectal nodule.

A fairly well-developed stroma of fibrous tissue was present, giving to the cut section of the tumour a lobulated appearance. The free surface of the growth was covered with a fibrinous exudate except at the upper and lower extremities, where the rectal mucosa and the squamous epithelium of the anal canal were intact for a short distance.

The extra-rectal nodule was made up almost entirely of an epithelial-like type of cell. The stroma was very scanty and the nodule was much more homogeneous in structure than the primary tumour. The cells were polyhedral in shape with abundant cytoplasm (*Fig.* 51). The nucleus was large and vesicular. Pigment was more abundant than in the primary tumour and was both extra-cellular and intra-cellular in position. The particles of pigment varied in size from fine powdery granules to large coarse deposits. The contrast between this epithelial

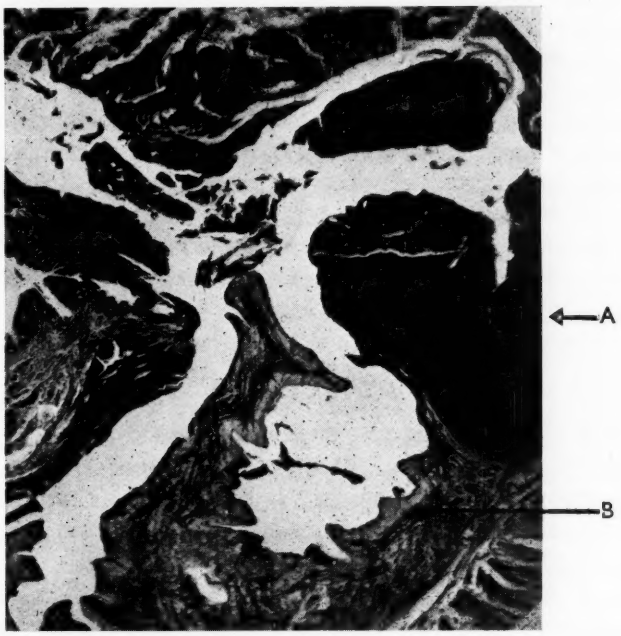


FIG. 55.—Low-power photomicrograph at the lower end of the tumour. On the right it can be seen that the tumour tissue (A) is in direct contact with the squamous epithelium of the anal canal (B). (× 5.)

type of cell and the sarcoma-like cell of the primary tumour is very striking, and this polymorphism of the cells of the melanomata is partly responsible for the difference of opinion as to their histogenesis.

The lymphatic glands showed pigmentation to the naked eye in nine out of ten cases, as already explained. On microscopical examination the pigmented glands all showed replacement of the lymphoid tissue to a greater or lesser degree by large cells, many of which were loaded with pigment granules. It is not an uncommon finding to have such glands described as malignant, to be the seat, in fact, of metastatic tumour cell deposit. Harding and Passey, working with a melanoma of the mouse, which they were able to transplant with success, were never able to obtain successful grafts from the pigmented glands which accompanied these tumours. They conclude, on this and other grounds, that the large pigment-bearing

cells in the glands were not truly tumour cells. We also have concluded that the cells containing pigment in the glands were not indicative of metastatic deposit but of transference or ingestion of pigment by phagocytic cells. The importance of this view with regard to prognosis in such a case is obvious.

In one gland, No. 4 on the chart, the lymphoid tissue had been entirely replaced by these pigment-carrying cells except for a narrow strip at the surface of the gland (*Fig.* 52). In the others, with one exception, there was a varying degree of replacement. The exception was No. 10, which lay on the anterior aspect of the rectum, away from the main stream of the lymphatic return. Where only a few melanophores were present, these were mostly found at the peripheral part of the gland, as if they had just entered from the afferent lymphatics. The assumption is that the pigment which is liberated by the breaking down of malignant cells in the primary tumour is ingested by the phagocytic pigment carriers and conveyed to the regional lymphatic glands. Some of the melanophores were so packed with pigment granules that the nucleus was almost invisible (*Fig.* 53).

A number of large microscopical sections of the tumour and the extra-rectal nodule were cut in the sagittal plane (*Fig.* 54). A close examination of these does not justify a dogmatic statement as to the origin of the tumour, but it was quite evident that the tumour tissue came into extremely close relationship with the squamous epithelium of the anal canal, as shown in *Fig.* 55. The tumour can be seen to lie immediately underneath the modified skin which lines the lower part of the canal. This fact is of importance in relation to the histogenesis of the tumour.

DISCUSSION

This case may be regarded as a typical illustration of melanoma of the rectum. Clinically, the symptoms are similar to those of adenocarcinoma of the rectum, and the condition will usually be mistaken for that lesion. Blackish discharge on the examining finger, however, might arouse suspicion as to the nature of the tumour. If the speculum is introduced, the dark colour of the growth will suggest its nature. In some cases there has been pigmentation of the anal orifice, and this appearance has sometimes been confused with thrombotic external hæmorrhoids. The tumour in the present case was sessile, but in many of the recorded cases it has been pedunculated—33 per cent (Loques)—and may even present at the anus as a polypus. A melanoma bulges into the lumen of the bowel; it shows no tendency to spread in an annular fashion, causing stenosis, as is frequently the case in adenocarcinoma. If it obstructs the bowel, as it may do in the long run, it does so by a process of occlusion and not of stenosis. It resembles in this respect a sarcoma of the rectum.

Origin of the Tumour.—Much discussion has taken place as to the essential nature of the melanomata, and the custom of naming these tumours according to the form and configuration of their cells—as in the use of the terms 'melano-sarcoma', '-carcinoma', '-endothelioma', '-perithelioma', and '-fibro-sarcoma'—was formerly, and to a certain extent still is, a cause of confusion. In one part of our specimen the appearances were those of 'melano-sarcoma', while in another they were those of 'melano-carcinoma'; it is evident, therefore, that such terms cannot be applied with real consistency to this type of tumour. Most English

writers follow the recommendation of Ewing and employ the term *melanoma*, to emphasize the specific character of the tumour and its cells of origin.

Ewing summarizes the various theories as to the origin of the melanomata. It is to be noted that he uses the term *chromatophore* in the sense that we, following the recommendation of Dawson, have used the term *melanoblast*, the pigment-producing tumour cell. He states that they arise either: (1) Exclusively from mesoblastic chromatophores (melanoblasts); (2) Exclusively from epithelial cells and epithelial chromatophores; (3) From nævus cells in the skin and mesoblastic chromatophores in choroid and meninges; (4) From endothelial cells of blood- and lymph-vessels, or of nerve-trunks; (5) From chromatophores, tactile corpuscles, and nerve-cells forming the end-apparatus of the cutaneous sensory nerves (Masson). Masson has brought forward very strong evidence in support of his view. Dawson, in his paper on the melanomata, expresses his belief that the basal cells of the epidermis are the true pigment producers; he traces the origin of quiescent nævus cells from the basal epithelium by a process of emigration and depigmentation; he derives all the histological varieties of cutaneous melanomata from the original basal cells; uveal melanomata, he states, are embryologically derived from the pigmented retinal epithelium; and melanomata of adrenal and other deep organs from pigmented neuro-epithelium, postulating a possible dispersion of ectodermal pigmented cells along the paths of out-growing nerves.

We do not feel competent to discuss a subject presenting such very great histological difficulties, but we should like to emphasize the fact that melanoma of the rectum almost always occurs low down, either in contact with the epidermal portion of the anal canal or close to it. Chalier and Bonnet found that in exceptional cases it might be as much as 5 to 6 cm. above the anus, i.e., 1 to 2·5 cm. above the anal canal, but even at this level the tumour may have been related to displaced portions of epidermis. The almost uniformly low position and the exceeding rarity of melanomata in other parts of the alimentary canal suggest that it probably arises from the skin of the anal canal. Chalier and Bonnet, after a most exact histological examination of their own case and a careful analysis of the 63 cases which they had collected, came to the conclusion that "primary melanotic tumours of the ano-rectal region are cutaneous epitheliomata from the anal canal, capable clinically of becoming tumours of the rectum by progressive infiltration along the length of the submucosa upwards". These writers believed that they were able to trace the exact point of departure of the tumour from the Malpighian epithelium of the anal canal. We were not able to do this in our specimen, but we observed that the tumour tissue came into the closest relationship to the squamous epithelium of the lower part of the anal canal. The evidence afforded by a study of melanoma of the rectum seems to favour the origin of this tumour from skin; we refrain from discussing whether it is more likely to arise from the basal cells of the epithelium (Dawson) or from the end apparatus of sensory nerves (Masson).

Spread of the Tumour.—Melanoma of the ano-rectal region invades the submucosa and shows a pronounced tendency to grow upwards into the rectum, which is presumably the direction of least resistance. It shows no special tendency to spread in a circular manner, but commonly bulges into the lumen of the bowel. At the same time it spreads deeply, and in our case has spread through the muscularis and invaded the perirectal fat. The layer of fat is enclosed in the fascial sheath of

the rectum, and this fibrous membrane, as happens in adenocarcinoma of the rectum, appeared to have contained or limited the spread of the growth. In the rectum, as elsewhere, melanotic tumours may be exceedingly malignant and may become generalized at an early stage, spread taking place both by blood-stream and by lymphatics. Secondaries have been found in almost every organ in the body, the liver being the most common site. Enlargement of the inguinal glands may be an early indication of metastasis. In our case there were one or two points which seemed to suggest a comparatively low degree of malignancy. There was no evidence of spread to lymphatic glands and no recognizable sign of spread to the liver. Although the tumour had spread through the muscularis, the extra-rectal growth presented appearances which might be interpreted as indicative of a lesser degree of malignancy than the primary growth. Harding and Passey have shown that the more malignant the melanoma the less pigment does it contain. The extra-rectal nodule showed more abundant pigment than the primary growth, and, its cells being large and epithelial in type, it might be considered to show a lesser degree of metaplasia. Sir David Wilkie has kindly supplied us with the notes of a case which are of interest in this respect. The patient, an elderly female, was operated on on April 29, 1933, and a papillomatous tumour of the rectum was removed by local excision; this proved on subsequent microscopical examination to be malignant. Two nodules were present in the neighbourhood, one of which was outside the wall of the bowel. It was felt that a radical operation was unlikely to be successful, and radium needles were inserted on May 15, 1933, after removal of the coccyx. The patient returned in June, 1934, with a recurrence of her symptoms. A small tumour was present in the wall of the rectum, which was found on microscopical examination to be a melanoma. The patient was again treated by radium. She returned in March, 1935, with extensive local recurrence in the rectal wall and peri-rectal tissues but without any evidence of general dissemination. A large dose of radium was again inserted extra-rectally. A bad prognosis was given, but in January, 1936, the patient reported in good health with no rectal trouble and having gained 18 lb. in weight. This case shows that dissemination of the tumour may be delayed for a long time, and also that these tumours may be very radio-sensitive, which lends support to the theory of baso-squamous epithelial origin. It is evident, therefore, that, where a radical operation is feasible, it must in some cases afford a reasonable prospect of success. However, until a large number of cases of melanoma of the rectum have been recorded and followed up it will not be possible to form an accurate estimate of the degree of malignancy of this type of tumour.

We have already referred to the misleading appearance of the lymphatic glands in this case. The pigmentation on naked-eye examination and the infiltration microscopically with large phagocytic pigment-carrying cells gave a false impression of malignant invasion.

CONCLUSIONS

1. The melanoma of the rectum is derived from the stratified squamous epithelial portion of the anal canal.

2. Regional lymph nodes which contain pigmented cells are not necessarily the seat of metastasis.

We desire to express our thanks to Sir David Wilkie for his advice and encouragement and for the facilities which he has given us in the Department of Surgical Research Laboratory. We are also much indebted to Colonel W. F. Harvey for his kindly interest in this paper. We wish to thank Mr. C. Shepley for the great care and skill with which he has executed the coloured illustrations.

BIBLIOGRAPHY

DAWSON, *Edin. Med. Jour.*, 1925, xxxii, 509.
CHALIER and BONNET, *Rev. de Chir.*, 1912, xlvi, 914.
CHURCHMAN, *Amer. Jour. Med. Sci.*, 1918, clv, 639.
KRAKER, *Trans. Amer. Proc. Soc.*, 1925, 102.
MCGUIRE and LEAHY, *Bull. Buffalo Gen. Hosp.*, 1924, ii, 85.
GOLDBLATT, *Jour. Amer. Med. Assoc.*, 1925, lxxxiv, 1986.
ALLAN, *Trans. Amer. Proc. Soc.*, 1931, xxxii, 31.
KALLET and SALTZSTEIN, *Arch. of Surg.*, 1933, xxvi, 633.
LANDMAN, *Trans. Amer. Proc. Soc.*, 1933, xxxiv, 65.
GERRITZEN, *Arch. f. klin. Chir.*, 1933, clxxviii, 400.
MARINO, *Jour. Amer. Med. Assoc.*, 1934, cii, 203.
INGLE, *Indian Med. Gaz.*, 1935, lxx, 266.
WOOD and WILKIE, *Edin. Med. Jour.*, 1933, xl, 321.
HARDING and PASSEY, *Jour. Pathol. and Bacteriol.*, 1930, xxxiii, 417.
EWING, *Neoplastic Diseases*, 3rd. ed., 1928.
MASSON, *Ann. d'Anat. pathol.*, 1926, iii, 417.

RADICAL EXCISION OF MALIGNANT TUMOURS IN THE ETHMOID-MAXILLARY REGION

By W. H. GRAHAM JESSOP
ASSISTANT, SURGICAL UNIT, UNIVERSITY COLLEGE HOSPITAL, LONDON

An opportunity occurred recently of examining a patient who was treated twelve years ago by a radical operation for an extensive malignant tumour in the left maxillary and ethmoidal regions. It is the purpose of the present paper to describe the operation employed in this case and to show that such an extensive procedure may have a limited but real field of usefulness.

CASE REPORT

The patient was an agricultural labourer, aged 44, who was admitted to hospital on Aug. 17, 1923, complaining that the left side of his nose had been blocked for the previous three months. Shortly after he noticed this, the left side of his face began to swell. During the first six weeks he had experienced pain at the back of the left eye, but this had subsequently disappeared.

A B

FIG. 56.—A, Appearance of the patient at the present time; B, The eye-shade removed. The sphenoidal sinus is visible at the back of the orbit.

On admission his general condition was good. There was a marked swelling of the left side of the face. The skin of the cheek was red, œdematous, and fixed to the maxilla, and on palpation areas of fluctuation were found. The eyeball was considerably proptosed, but the palpebral fissure was occluded by a marked œdema of the eyelids, especially of the lower. The left nostril was completely blocked by a mass arising from the lateral wall. The left half of the hard palate was depressed into the mouth, forming a rounded, fluctuating swelling, and there was a hard, smooth mass to the outer side of the left upper alveolus. No enlarged lymphatic glands were felt in the neck.

An X-ray film showed irregular opacities in the left nasal fossa, left antrum, left frontal and left ethmoidal sinuses suggestive of malignant disease.

A biopsy from the mass in the nostril was performed and a section showed that the growth was an endothelioma.

On Oct. 17, 1923, a radical operation for the removal of the growth was performed by Mr. Wilfred Trotter. After a preliminary laryngotomy the anæsthetic was given through the laryngotomy tube and the pharynx carefully plugged. The whole tumour, together with a margin of normal tissue, was removed by the method described in detail below. During the operation the frontal and ethmoidal sinuses were opened and found to contain polypoid material. Recovery was uneventful and the patient left hospital six weeks later.

When seen on Dec. 12, 1935, over twelve years later, the patient was in excellent general health and spirits. There was no sign of local recurrence or metastsais. He wore an eye-shade to cover the defect in the base of the orbit, but otherwise the external deformity was minimal (*Fig.* 56). He stated that his acquaintances believed that the eye-shade concealed a 'weak' eye.

On inspection of the buccal cavity an oval recess 3 cm. by 2 cm. was visible at the site of the left upper alveolus and left half of the hard palate. The soft palate was intact, but somewhat drawn up on the left side. Through the gap in the facial scar the nasal septum was visible medially, and behind this the left sphenoidal sinus. Inferiorly the left half of the soft palate was visible with the nasopharynx above and behind and the tongue below and in front.

The patient had been perfectly well and free from pain since recovery from the immediate effects of the operation. A plastic operation for the closure of the naso-orbital defect has been suggested to him; but as he would still have to wear an eye-shade he is disinclined to bother about it.

THE OPERATION

The object of the operation is to remove intact the whole of the outer wall of the nose (*Figs.* 57, 58). The most inaccessible part of this mass of tissue is the lateral mass of the ethmoid, where experience shows that many of the growths of this region originate. The lateral mass of the ethmoid cannot be removed intact unless it is possible to sacrifice the contents of the orbit. In the advanced cases for which the operation is particularly designed, it is already clear from the proptosis and possibly chemosis that the normal position and function of the eye cannot be preserved or regained, and there is no objection to the approach through the orbit, which is an essential step in the extirpation of the ethmoid. Many of the growths originating in the lateral mass of the ethmoid involve the maxillary antrum comparatively late, and in these cases the hard palate may be preserved without risk.

The operation is definitely designed for advanced cases, and the surgeon should beware of concluding too readily that a given case is inoperable. Much of the apparently hopeless aspect of the case reported here was due to the severe secondary inflammation of the accessory sinuses. Spread of the disease to the tissues of the apex of the orbit and the foramina there is perhaps the commonest cause of failure in apparently hopeful cases.

The incision is of the Ferguson type, but enters the palpebral fissure at the inner canthus and leaves it at the outer canthus to be prolonged along the zygoma.

The whole of the soft tissues in the infero-lateral flap, if not involved in the growth, is raised from the bone and thrown outwards. In cases where involvement of the antrum is doubtful a small opening is made in the anterior surface of the maxilla and the condition of the cavity decided by inspection. The upper flap, including the upper eyelid, is next dissected up, the external angular process of the frontal divided, and the zygoma cut through, or, better, the middle portion removed. The outer wall of the orbit is now exposed and removed, giving free access to the orbital contents. The latter are carefully separated by working back to the apex of the orbit until the extreme posterior end of the inner wall is exposed. They are also cleared from the roof, remaining attached only to the lateral mass of the ethmoid and the maxilla with which they will be removed.

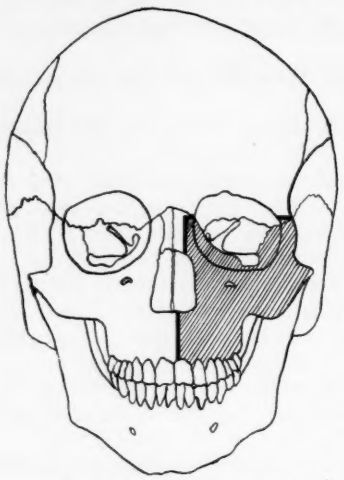


FIG. 57.—Outline of skull showing sites at which bone is divided. The portion to be excised is shaded.

If the antrum is invaded by the growth, the next step is to divide the alveolus and the hard palate close to the septum, subsequently separating off the soft palate. When the antrum is free the corresponding cut is made horizontally across the maxilla above the level of the hard palate, which is preserved.

After a saw cut has separated the nasal bone from the nasal process of the maxilla, the separation of the lateral mass of the ethmoid from the frontal can be undertaken. A chisel driven directly backwards at the level of the orbital roof effects this division, the frontal sinus being opened in the process. The mass which has thus been outlined is now gently levered loose from its remaining bony attachments posteriorly.

The excised parts, which should come away in a compact unbroken mass, include the maxilla, the lateral mass of the ethmoid, the malar bone, and the orbital contents, and on inspection of the inner surface the whole of the lateral wall of the nasal fossa is visible.

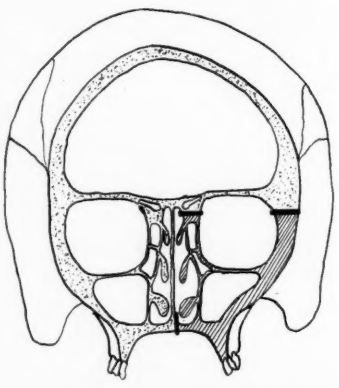


FIG. 58.—Coronal section of skull, diagrammatic, to show the relations of the divisions to the surrounding structures.

DISCUSSION

The present paper is intended to deal with only one limited part of the large topic of naso-maxillary malignant disease. The case described illustrates, however, certain of the more general aspects of the subject, which may therefore be briefly referred to.

Secondary suppuration and polypoid disease of the accessory sinuses is a well-known complication of malignant disease in the region under discussion. It was very severe in the case described, causing profuse purulent discharge and much brawny swelling of the cheek. No doubt it is due to obstruction of the sinuses

by the growth. It is an important complication in two respects. First, it may so much aggravate the severity of the picture produced by the tumour itself as to make an operable case seem utterly hopeless. Secondly, it adds greatly to the difficulties of applying radiotherapy to these cases. There seems, moreover, little doubt that the presence of secondary inflammation favours rather than discourages the spread of the primary neoplasm.

The site of origin of the tumour is of the first importance in designing the treatment of any given case. In former times it was apt to be assumed much too readily that growths of the class under consideration have their origin in the maxilla. The result was that they were treated by a formal excision of the upper jaw without the least prospect, in many cases, of any good being done. More exact methods have shown that while in many cases the growth does begin in the maxillary antrum, or penetrate to it from the alveolus, probably an equal number begins in the outer wall of the nose in relation to the lateral mass of the ethmoid. It is for dealing with these latter cases that the operation described above is suitable. The precise diagnosis of the site of origin of the tumour is therefore very important, though not always attainable. In the present case the early blockage of the nostril, and the pain behind the eye, were regarded as pointing to a growth arising in the lateral mass of the ethmoid.

It may be interesting to quote the opinion of three recent authors on the problem of treating maxillary-ethmoidal malignant growths. Quick[1] (1930) states that it is doubtful if a radical operation conforming to general surgical principles for the treatment of malignant disease is possible in this region. Davis[2] (1934) concludes that the difficulty of early diagnosis and the inaccessibility of ethmoidal carcinoma makes it doubtful whether such a mutilating operation is worth while. Harmer[3] (1935), in an able presentation of the case for combined partial excision and radiotherapy, reaches the conclusion that approach should never be made through the skin. It may be agreed that in highly malignant radio-sensitive growths complete local regression is obtainable by the combined method, but is too commonly followed by death from distant metastases. In radio-resistant types the course following any type of irradiation is too frequently persistence of the growth accompanied by severe and intractable pain.

It seems clear that in cases conforming to the indications laid down the method of radical extirpation here described has certain advantages over other procedures. Suitable cases must necessarily be few, and their most important characteristics will be that the disease is in a fairly advanced stage and that it shows evidence of having originated in the outer wall of the nose.

SUMMARY

1. A case of successful radical excision of an extensive malignant tumour in the maxillary and ethmoidal regions is described.

2. The operative procedure is described.

3. An attempt is made to define the conditions in which such an operation may be the best treatment.

REFERENCES

[1] QUICK, D., *Radiology*, 1930, xiv, 191.
[2] DAVIS, E. D. D., *Brit. Med. Jour.*, 1934, i, 53.
[3] HARMER, W. D., *Lancet*, 1935, i, 129.

SPONDYLOLISTHESIS

By JAMES A. JENKINS, New Zealand

In recent numbers of the British Journal of Surgery three papers have appeared on this interesting condition. Shore[1] describes this deformity as found in the Bantu races of South Africa, and the developmental anomalies which predispose to the condition are discussed. Brailsford[2] gives a comprehensive review of the subject and reports several cases. He concludes with the following statement: "A study of the specimens and radiographs in my cases negatives the suggested possibility of manipulative reduction of the displacement." Capener[3] similarly reviews the literature and bases his paper on the study of three cases. In discussing the surgical treatment he points out that while bone-grafts undoubtedly assist many patients, they do not, from the mechanical point of view, possess great efficiency. "Tremendous forces are brought to bear upon the graft, which is badly placed for the prevention of slipping." "The ideal operation would be an anterior bone-graft so placed as to fix the body of the fifth lumbar vertebra to the sacrum and forming a buttress or some form of antero-posterior fixation of the two halves of the divided vertebra. The technical difficulties of such a procedure, however, preclude their trial."*

In view of the fact that nothing can be added to the developmental, clinical, and radiological aspects discussed in the papers that have recently appeared, and as complete bibliographies are appended thereto, repetition would be wearisome. No paper has indicated a course of treatment, either conservative or surgical, that can arouse any enthusiasm for end-results.

In the case here reported a gross deformity was reduced, and the method of fixation followed was that suggested by Capener as the ideal operation though impracticable on account of technical difficulties.

The deformity in an advanced case of spondylolisthesis is marked and the disability very great. Pessimism both as regards reduction of the deformity and means of adequate fixation permeates all papers. Jackets and various supports are suggested, but it is obvious that these cannot be effective if the patient is to lead an active life. Bone-grafts on the Albee principle are mentioned and the obvious mechanical inefficiency of these is referred to. The fact that the division in the vertebra is through the arch, and the graft therefore does not directly fix the bone that is slipping, makes it apparent that only imperfect fixation of the body can be achieved by this indirect means.

A graft consolidating the third and fourth lumbar vertebra together with the separated posterior arch of the fifth lumbar vertebra and the sacrum must do something towards stabilization of this region, but the tremendous stress when there is complete overlapping of the fifth lumbar vertebra on the first sacral is so great that the bad results reported can be readily understood.

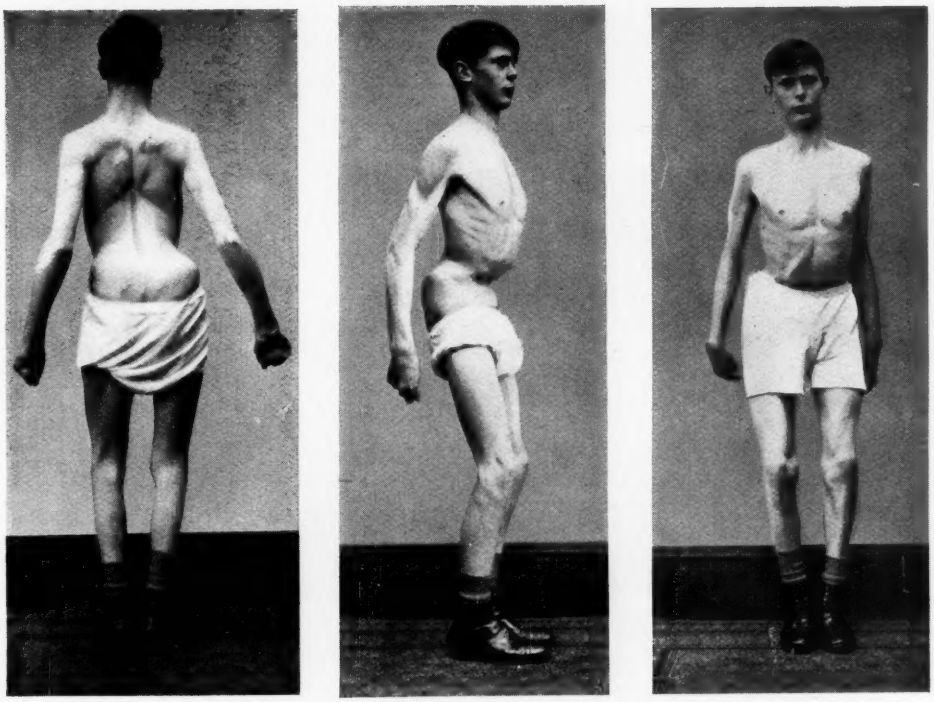
---

* A case of spondylolisthesis in a boy of 14, of traumatic origin, was reported by Burns in *The Lancet*, June 10, 1933. He was treated by driving an autogenous tibial graft from the abdominal aspect of the 5th lumbar vertebra into the sacrum, with success (Ed.).

Albee states that the fourth and fifth lumbar vertebræ tend to pull themselves away from the graft in an antero-posterior direction. Brailsford quotes Turner and Tchirkin: "It is doubtful whether operative measures can aid conservative treatment in alleviating the symptoms and arresting the progress of the ailment." He also goes on to say that a study of the specimens and radiographs negatives the suggested possibility of manipulative reduction of the displacement.

CASE REPORT

A male, aged 16 years, complained of a 'bad hip'. Deformity commenced a year previously and had become progressively worse. He was easily tired, much handicapped by the posture he was forced into, and suffered pain in the lower part of the back after exercise. He could remember no previous accident, but had played the usual school games and suffered the customary injuries.



FIGS. 59–61.—Photographs showing the deformity prior to operation.

Examination showed the deformity characteristic of advanced spondylolisthesis. The posterior projection of the pelvic girdle, the forward direction of the lower lumbar spine, the shortening and forward displacement of the lumbar spine, and the compensatory lordosis, are well shown in *Figs.* 59–61.

A radiograph (*Fig.* 62) taken when the patient was first seen showed a complete displacement of the body of the fifth lumbar vertebra, its lower border lying opposite the second sacral vertebra. After admission to hospital, reduction was attempted by continuous extension. The pelvis was suspended by a sling to an overhead frame. By means of Sinclair's glue extension was applied to both legs and pelvis at the one end and to the chest at the other: 60 to 70 lb. weight were applied. The patient was encouraged to manipulate his own pelvis at frequent intervals by catching the iliac crests in his hands and forcibly pushing them away. On the fourth day he was conscious of a grating noise and a sense of change of position, and

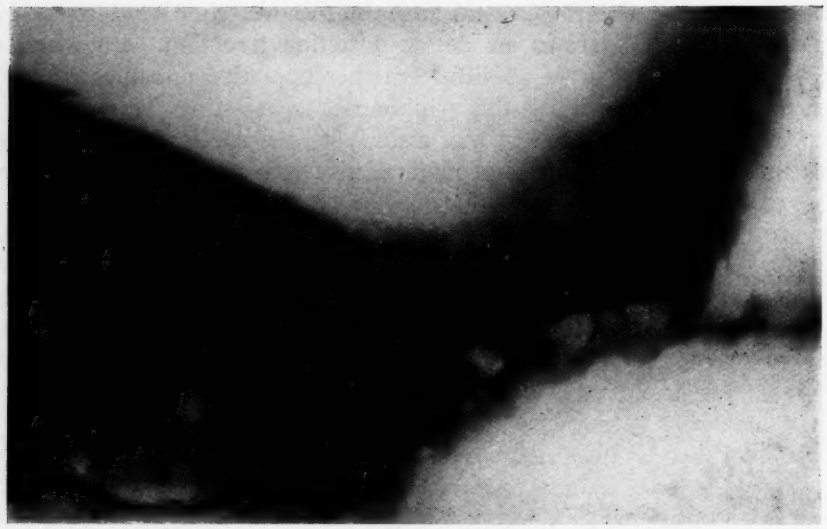
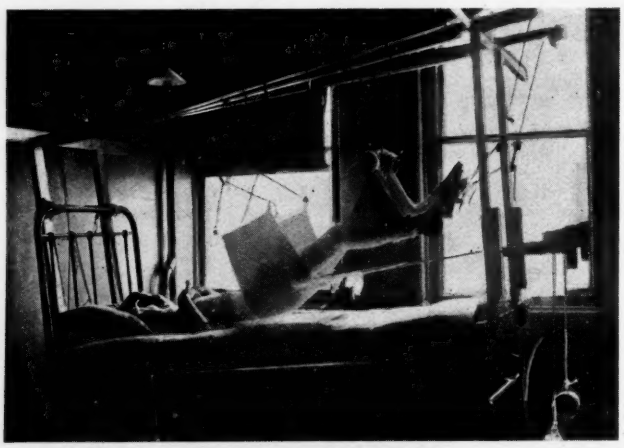
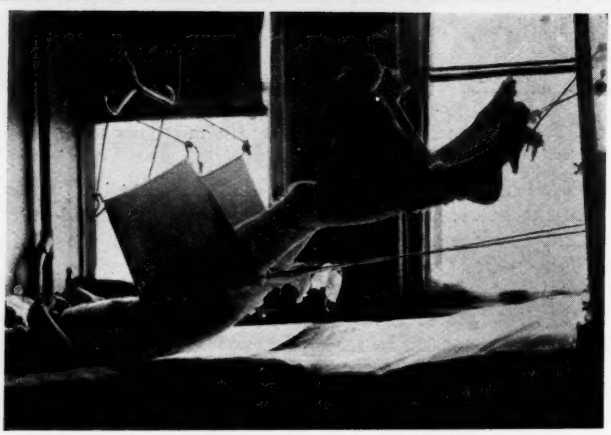


FIG. 62.—X-ray of patient when first seen (Sept. 29, 1934).





FIGS. 63, 64.—Showing the position after reduction and the way in which extension was applied.

examination showed that the deformity had largely disappeared. *Figs.* 63 and 64 show the position after reduction and the way in which the extension was applied. An X-ray (*Fig.* 65) taken at this time showed that the lower surface of the fifth lumbar vertebra was now resting on the upper surface of the sacrum, but there was about half a vertebra overlap in an anterior

FIG. 65.—X-ray taken on Nov. 26, 1934.

posterior direction. No further improvement was achieved by manipulation and extension, though it was carried out for a further period of two weeks.

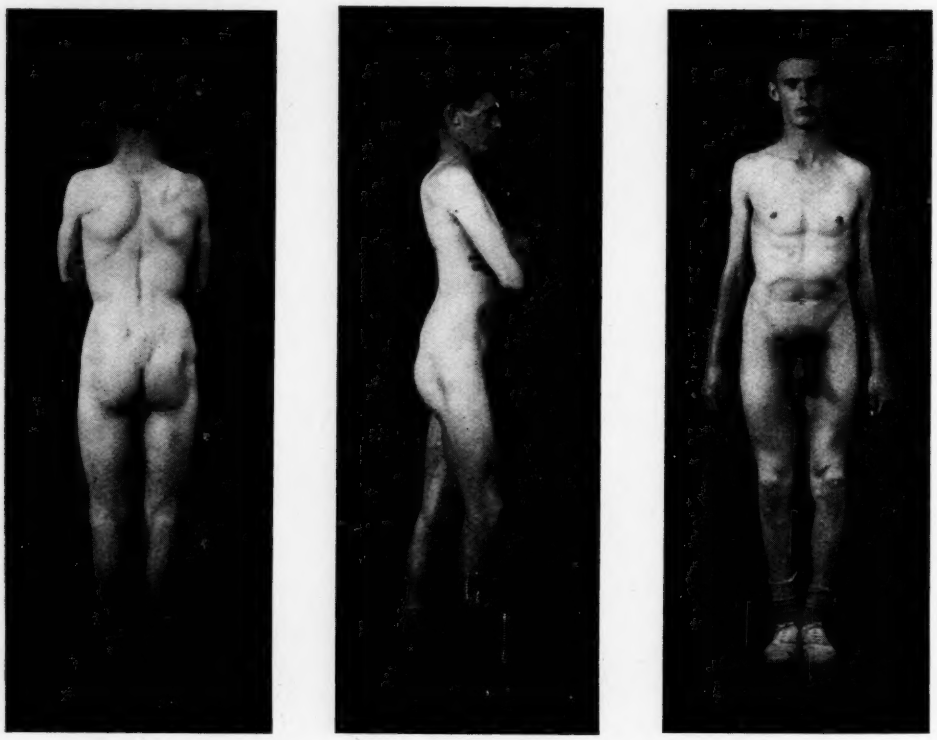
OPERATION (Dec. 10, 1934).—The anæsthetic was rectal paraldehyde followed by novocain spinal anæsthetic supplemented by light ether anæsthesia. The abdomen was opened

FIG. 66.—X-ray taken after operation (Feb. 8, 1935).

by a right paramedian incision extending from above the umbilicus to a little above the pubes. The intestine was packed off and the sacral promontory exposed. The peritoneum over the promontory was incised for 3 in and the nerve plexus, left common iliac vein, and bifurcation

of the aorta were defined. The anterior long ligament of the spinal column was divided for ¾ in. over the anterior aspect of the fifth vertebra. Slight oozing was encountered here, and this was a little troublesome throughout the operation. With a bone-drill $\frac{5}{12}$ in. in diameter a hole was made through the body of the fifth lumbar vertebra into the anterior aspect of the first sacral vertebra for a distance of a little more than 2 in. A bone-graft cut from the tibia with an Albee saw was driven into the track made for it, a very tight and firm fit resulting. It was not found possible, as was hoped, to close the anterior longitudinal ligament over the graft. Horsley's wax was used to stop slight bone oozing. The posterior parietal peritoneum was closed and the abdomen sutured in the usual way. The operation took an hour and a quarter and the patient's condition gave no cause for anxiety at its termination. Apart from a pulse ranging from 110 to 120 for a few days convalescence was uneventful.

SUBSEQUENT PROGRESS.—Radiographs taken on Feb. 8 (*Fig.* 66) showed the graft pinning the fifth lumbar vertebra to the anterior aspect of the body of the first sacral vertebra. A plaster spinal support was applied and the patient allowed up. The support was removed three



FIGS. 67–69.—Photographs of the patient a year after operation.

months later, and since then ordinary activities have been carried on without the use of any support.

Photographs (*Figs.* 67–69) of the patient's condition a year after the operation show that the deformity has not been completely reduced but that very marked improvement has taken place. He can now walk without difficulty and pain.

Whether the surgical procedure here outlined will become the method of choice in the treatment of these cases can only be known when a large series has been done. The strain on the graft, though considerable, is not as great as would appear, as the inability of the body of the fifth lumbar vertebra to slip forward

ensures that most of the weight is still transmitted through the upper surface of the body of the first piece of the sacrum. The operation presents difficulties and risks much greater than those encountered in a posterior graft of the Albee type and results will have to be proportionately better if it is to be adopted in the treatment of this very difficult condition.

In a future case, in addition to the graft, an attempt will be made to increase the bone union between the fifth lumbar vertebra and the sacrum by elevating bone-flaps and getting direct bone union between the vertebræ themselves.

SUMMARY

1. A method of reduction of the deformity in spondylolisthesis is described.

2. Fixation by a bone-graft through the bodies of the vertebra has proved an efficient means of stabilization.

REFERENCES

[1] SHORE, *Brit. Jour. Surg.*, 1929, xvi, 431.
[2] BRAILSFORD, *Ibid.*, 562.
[3] CAPENER, *Ibid.*, 1932, xix, 374.

THE CLINICAL MANIFESTATIONS OF THE SPREAD OF CARCINOMA OF THE ŒSOPHAGUS OBSERVED DURING LIFE

By JOHN E. G. McGIBBON, Liverpool

The following observations are based on a series of 100 personal cases of carcinoma of the œsophagus which have been examined and treated during the past seven years. Post-cricoid and gastro-œsophageal neoplasms have not been included, and the series is confined entirely to growths affecting the œsophagus proper.

The analysis of the case histories was instigated by the considerable literature which has accumulated within recent years on the actual, experimental, and theoretical methods of surgical removal of malignant tumours of the gullet, and the feature that became manifest from this analysis was the large number of cases which showed evidence of spread of the growth at or about the time of the first examination.

Apart from the inaccessibility of the œsophagus, its intimate relationship to other vital structures, and its poor response to surgical trauma, the possibility of radical surgical treatment of œsophageal neoplasms would appear to depend upon two debatable but related factors—namely, early diagnosis and virulence of the growth.

DIAGNOSIS

Diagnosis invariably is late, as it is dependent upon the onset of symptoms; and it is well recognized that unfortunately the early symptomatology of cancer of the œsophagus is vague, and its manifestations are apt to be regarded as trivial and unimportant. There is of necessity a latent period between the earliest carcinomatous formation and the patient's awareness of something amiss, and the duration of this period at present cannot be estimated, although serological methods may in the near future yield information on this point.

The most common complaint that causes patients to seek advice is difficulty in swallowing. Of the present series of 100 cases, 91 came complaining of dysphagia, 4 of pain, 1 of hoarseness, 2 of impaction of foreign body, 1 of glands in the neck, and 1 of hiccup. Watson[1] states that of a series of 506 patients suffering from carcinoma of the œsophagus who were admitted to the Memorial Hospital, New York, in 64 per cent dysphagia was the first symptom; and Chevalier Jackson[2] records that of 671 such cases, in 649 difficulty in swallowing first drew attention to the disease. The dysphagia is caused by an actual narrowing of the œsophageal lumen by tumour formation with or without a superadded muscular spasm, and so by the time this symptom appears the growth must have extended into the lumen sufficiently to interfere with the passage of food down the gullet, and it is reasonable to assume that it has spread also in other directions. As a rule the onset and

progress of the dysphagia is insidious or intermittent, and patients unconsciously masticate more thoroughly, take soft foods, and aid deglution by copious draughts of fluids. Occasionally attention is drawn suddenly to the œsophageal lesion by the impaction of a bolus of food in the strictured area of the œsophagus; this occurred in two cases of the present series. It is surprising through what a small channel any well-masticated food may be swallowed without apparent difficulty; thus Jackson[3] states that a lumen of 5 mm. affords quite an efficient food conduit.

Even when dysphagia has become well established, patients fail to obtain expert advice, and valuable time is lost. Responsibility for this loss is not due entirely to ignorance, carelessness, or fear on the part of the patient, as not infrequently they have been treated by their medical attendant for 'nervous spasm' or 'dyspepsia' for two or three months before the nature and gravity of the complaint was realized. Jackson[2] records that of 110 cases of œsophagoscopically proven malignant disease of the œsophagus, in 87 an inferential diagnosis of neurotic condition had previously been made.

Of the present series, the average duration of symptoms before help was sought was 3·4 months—the longest period was 11 months, and the shortest was 14 days. Abel[4] states that the average duration of symptoms before a case comes to the surgeon is from 6 to 8 months. In Souttar's[5] series the average time lost in this manner was 5·2 months. Broders and Vinson[6] estimate this period as 7 months, and Watson[1] as 5·8 months. The average period between the onset of symptoms and diagnosis, therefore, is almost 6 months, and this may be even a month or two longer, as patients are notoriously inaccurate when recounting the duration of their symptoms.

VIRULENCE

In regard to the virulence of œsophageal carcinomata, opinions differ very widely, and Grey Turner,[37] after long observation, has stated 'that in the œsophagus as in other parts of the body, malignant disease varies in type and in behaviour.' Several observers regard the growths as relatively benign, and Abel,[7] who reviewed the findings of numerous investigators, concluded that at least one out of every four cases is operable by radical surgical methods—even at the time of death. Other writers (Wright,[8] Musgrave Woodman,[9] and Chevalier Jackson[2]) consider the tumours to be of low virulence so long as they are confined to the œsophagus itself, but that once they transgress these boundaries their growth is rapid and destructive. Lastly, there is a growing volume of opinion that œsophageal neoplasms are very virulent lesions. Vinson,[10] reviewing 1000 cases of carcinoma of the œsophagus which were seen at the Mayo Clinic during a ten-year period, concludes that the condition is very highly malignant, as indicated by its rapidly progressive clinical course. Presumptive evidence of their virulence is afforded by the results of a recent investigation by Roberts,[11] who analysed the post-mortem findings of a large number of patients who had died from malignant disease of various regions. He found that the gravity of the case bore a definite relationship to the proximity of the tumour to the mediastinal lymph-glands, particularly to the tracheo-bronchial group.

The rapidly killing properties of malignant œsophageal tumours may be gauged by the following observations. Watson,[1] in a series of 208 cases, found that the

average length of life after the appearance of the first symptoms was 10·5 months, and after application to hospital 4·83 months. From Souttar's[5] published cases an analysis shows that the average survival period from the onset of the first symptoms was 10·5 months, and that from the time of application to hospital was 4·7 months. Of the present series, only 57 cases have been followed to death up to date, and of these the average duration of life from the first symptoms was 9·8 months, and that from the time of application to hospital was 5·9 months. Clayton[12] estimates the average length of life from the appearance of the first symptoms to be 7 months. Chevalier Jackson,[2] on the other hand, states that if patients suffering from carcinoma of the œsophagus have an abundance of water and a full allowance of a well-balanced diet, they will survive for at least 2 years—several have been known to live for 5 years, and one for 6 years.

Post-mortem examination of patients who have died from carcinoma of the œsophagus frequently reveals widespread local and distant extension; at other times no macroscopic secondaries are manifest. Souttar[5] and Watson[1] state that the virulence of the growths is such that they frequently kill the patient before there is time for the formation of secondary tumours. This statement is borne out by the observation that in a series of 70 autopsies on patients who died from carcinoma of the œsophagus, Helsley[13] found that those who had no demonstrable metastases had had symptoms of an average duration of 4·5 months only, which is about half the average survival time from the onset of symptoms for all cases. Ewing[14] is of the opinion that such growths form early and extensive metastases, and Illingworth and Dick[15] state that fully 50 per cent of such cases which come to post-mortem show spread by the lymph-vascular system. Of the present series, only 13 cases have come to autopsy, and of these 10 showed metastases. Clayton[12] states that, of 41 post-mortem examinations, in 21 he found metastases below the diaphragm, and in 11 of these the liver was involved. Souttar[5] reports macroscopic secondary growths in 8 cases out of 18; Watson[1] in 14 out of 27; and Broders and Vinson[6] in 27 out of 42 post-mortem examinations.

Histologically carcinomata of the œsophagus are mostly of a highly malignant type when graded by Broders' method. *Table I* shows the findings of three groups of observers :—

Table I.—Relative Frequency of Different Grades of Œsophageal Carcinoma

Observer	No. of Cases	Grade I	Grade II	Grade III	Grade IV
Broders and Vinson[6]	207	—	16	95	96
Watson[1] ..	202	15	148	39	—
Clayton[12] ..	39	3	15	12	9
Percentages	—	4%	40%	32·5%	23·5%

It is of interest to note also that Cronin Lowe[16] has found by his modification of the Bendien test that patients with carcinoma of the œsophagus show a more

markedly positive serological reaction than do patients developing carcinoma elsewhere; and he has kindly allowed me to quote his unpublished data :—

12 *Carcinoma of Breast.*—Early cases, clinically and surgically diagnosed as such. Average reaction, 27·5.
12 *Carcinoma of Cervix Uteri.*—Early cases. Average reaction, 27·5.
12 *Carcinoma of Rectum and Colon.*—Early cases, many of them removable by excision. Average reaction, 27.
12 *Carcinoma of Stomach.*—Early cases, several treated by gastrectomy. Average reaction, 26.
12 *Carcinoma of Œsophagus.*—Average reaction, 24.

From these observations he suggests that carcinoma of the œsophagus may insidiously develop to a greater degree of malignancy before clinical diagnosis is obvious than is the case in the development of carcinoma in the other areas referred to in the table; his findings also emphasize the fact of late diagnosis.

MODES OF SPREAD

Of the 100 cases under review, 29 showed definite clinical evidence of extension in or beyond the œsophagus at or about the time of the first examination. The possible modes of spread of œsophageal carcinoma are by one or more of the following routes: (1) Direct extension; (2) Lymphatic permeation and embolism; (3) Bloodstream; (4) Implantation. Abel[7] describes a further mode—by physiological propulsion—but this can be included under the heading of implantation for the purpose of these notes.

DIRECT EXTENSION

Symptoms and signs of spread by direct extension will naturally depend upon the level of the growth. In its upper third the œsophagus is in close contact with the trachea, recurrent laryngeal nerves, carotid arteries, and pleuræ. In its middle third with the trachea, left bronchus, intercostal arteries, thoracic duct, hemiazygos veins, aortic arch, left subclavian artery, and right pleura. In its lower third with the left auricle, pericardium, descending thoracic aorta, and pleuræ. Thus the œsophagus in its upper two-thirds is in relation to many important structures (*Fig.* 70), and spread from growths situated in this locality is more likely to give rise to signs and symptoms than from those situated below the tracheal bifurcation.

Excluding post-cricoid and gastro-œsophageal growths, the most frequent site of œsophageal carcinoma is still undetermined. *Table II* gives the percentage location recorded by several observers, and it would appear from this analysis that a large proportion of growths occur in the middle third of the œsophagus.

Recurrent Laryngeal Nerve.—The most common manifest complication of spread in this series was caused by involvement of the recurrent laryngeal nerves. This finding is in accord with the observations of numerous writers, and it is of interest to note that such cases supplied the data on which Semon[21] based his law fifty years ago. Pressure on or destruction of the recurrent laryngeal nerve by actual growth or enlarged glands is characterized by hoarseness, aphonia, cough, dyspnœa, and paralysis of the vocal cord or cords when viewed by the laryngeal mirror. Hoarseness due to vocal-cord paralysis must be differentiated from the

hoarseness of a laryngitis caused by overflow of mucus, etc., into the larynx. This latter complication has been stressed by Chevalier Jackson,[3] and is mentioned later.

There were 13 patients of the present series who exhibited vocal-cord paralysis when they first applied for treatment. In 8 cases the left cord was affected, in 4 the right cord, and in 1 case both cords were immobile. Cases due to fixation of the arytenoid by post-cricoid or hypopharyngeal neoplasm have not been included

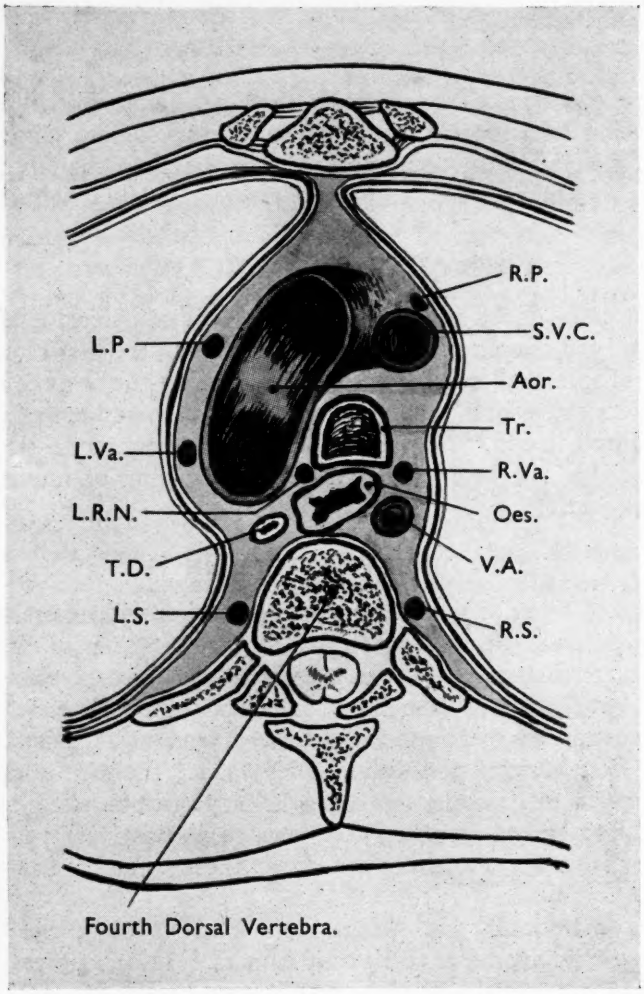


FIG. 70.—Relations of œsophagus at level of fourth dorsal vetebra. L.P. and R.P., Phrenic nerves: L.R.N., Left recurrent nerve; R. and L. Va., Vagi; R.S. and L.S., Sympathetic nerves; T.D., Thoracic duct; V.A., Azygos vein.

in this series. The paralysed cords when viewed by the laryngeal mirror were seen to be in the cadaveric position. In one patient with left-cord paralysis who later developed a right-cord palsy, it was possible to observe the sequence of abductor paralysis followed by the adoption of the cadaveric attitude.

The four patients who exhibited right-cord palsy were all males with neoplasms situated respectively at 11 in., 13 in., 13½ in., and 14 in. from the incisor teeth.

The growths in these four cases were far below the neighbourhood of the right recurrent nerve, and the paralyses were probably due to pressure on the nerve by enlarged lymphatic glands of the right paratracheal (recurrent) chain (*see Fig.* 75); consequently these cases are included under the heading of lymphatic spread, leaving 9 cases only which clinically showed evidence of recurrent nerve involvement due to direct spread from an œsophageal neoplasm.

Table II.—PERCENTAGE LOCATION OF ŒSOPHAGEAL CARCINOMA

OBSERVER	UPPER THIRD	MIDDLE THIRD	LOWER THIRD
	Per cent	Per cent	Per cent
Clayton[12] ..	10	56	34
Kraus[17]	18	36	46
Janeway and Green[17] ..	16	32	52
Watson[1]	19	27	54
Guisez[18]	34	46	20
Mackenzie[18] ..	13	50	37
Rawling[18] ..	10	80	10
Binnie[18]	16	32	52
Lamy[18] ..	10	36	54
Abel[7]	29	46	25
Souttar[5]	22	63	15
Cleminson and Monkhouse[19] ..	7	62	31
Klein[20]	14·6	46·7	27·2
Von Hacker[20] ..	12·3	48·8	26
Lotheissen[20] ..	13·3	48·8	37
Kraus-Ridder[20] ..	27	30	56
Present series ..	22	51	27
Mean percentage	**17**	**47**	**36**

The patient who exhibited bilateral vocal-cord palsy was the only one who gave a history of hoarseness *preceding* dysphagia:—

Case 1.—W. A. B., male, aged 60 years. Complained of hoarseness of four months' and dyspnœa of one month's duration. He stated that dysphagia had been present for fourteen days only. Examination showed that both vocal cords were fixed in the cadaveric position. Probably his left cord had become paralysed four months before coming to hospital, giving rise to hoarseness, and this was followed three months later by paralysis of the right cord, which caused the dyspnœa. This was an interesting case, as he had two malignant ulcers of the œsophagus, which are described and illustrated below (*see Fig.* 76).

Recurrent-nerve paralysis indicates extensive extra-œsophageal spread, which should definitely exclude any attempt at radical treatment. Only one patient of the present series with left-cord paralysis came to autopsy, and examination showed a small ulcer of the middle third of the œsophagus, whilst the entire mediastinum was filled with carcinomatous growth apparently invading all the neighbouring structures. Guisez,[22] from his wide experience, regards such cases as unsuitable even for palliation by radium, and he has stated[23] that 1 out of 5 of all cases of carcinoma of the œsophagus develop vocal-cord paralysis, and that when the growth is situated in the upper third paralysis occurs in 1 out of every 2 cases. He records 60 cases, of which 6 were bilateral. New and Chidley[24] have reported 34 cases of recurrent-nerve paralysis in patients suffering from carcinoma of the œsophagus. In their series the left cord was affected in 18 cases, the right in 13, and in 3 the paralysis was bilateral. Watson,[1] in his large series of 245 cases, reports 'hoarseness' as a symptom in 2 only, and Clayton[12] states that of a series of 41 autopsies, hoarseness had been complained of in 5 cases.

Phrenic Nerve.—It has been stated by several observers that a troublesome hiccup occurring in the course of carcinoma of the œsophagus is indicative of involvement of the phrenic nerve—this would probably be due to an irritative lesion. Abel[7] states that paralysis of the diaphragm and massive collapse of the lung may occur as secondary phenomena in the presence of phrenic nerve paralysis. Hiccup was the first symptom complained of by one patient of the present series:—

Case 2.—W. B. S., male, aged 62 years. Complained in June, 1934, of persistent hiccup, some giddiness, and nose bleeding; he made no complaint of dysphagia. His blood-pressure was 205/125, and he had some cardiac hypertrophy and arteriosclerosis. In August, following routine X-ray examination, Dr. J. H. Mather reported: 'The appearances are typical of early malignant disease of the œsophagus at level of 8th D.V. There is a slight hold-up of thick paste at the site of the growth, but fluids pass without difficulty.' Œsophagoscopy showed a small malignant ulcer 10¼ in. from the incisor teeth into which radon seeds were inserted. A snipping removed was reported by Dr. Howel Evans to be epitheliomatous. This was considered to be an early and fairly hopeful lesion, and the presence of extra-œsophageal spread was not anticipated as the significance of the hiccup was not recognized. The patient died five months later of auricular fibrillation. No autopsy was obtained.

Watson[1] records two cases of severe hiccup in his series.

Intercostal Nerves and Vertebræ.—The intercostal nerves are not infrequently affected either directly or as a sequel of carcinomatous extension of the vertebræ. Carcinoma of the œsophagus is essentially a painless disease, and when pain is present it is an indication of extra-œsophageal spread. Discomfort and fullness behind the sternum are common complaints, and odynphagia may be present in growths of the extreme upper end of the œsophagus, but pain occurring independently of swallowing in patients with tumours situated below this level is a symptom of serious import. Four patients of the present series sought advice because of severe pain—they complained also of dysphagia, but in all four cases this was a minor complaint.

Case 3.—A. B., female, aged 60 years. With a malignant ulcer in the upper third of her œsophagus complained of severe pain from which she had suffered for three months. X-ray examination showed a malignant stricture of the œsophagus with definite destruction of the body of the seventh cervical vertebra (*Fig.* 71).

Two male patients, aged 57 years and 63 years respectively, with middle-third growths experienced agonizing girdle pain. Both of these patients could swallow

comfortably after intubation, but it was found necessary to keep them under the influence of large doses of morphia to relieve their suffering. X-ray films of one of these patients showed involvement of the body of the eighth dorsal vertebra; no X-ray changes in the spinal column were manifest in the other patient.

The fourth case was that of a female patient suffering from carcinoma of the upper œsophagus with extension to the thyroid gland. She complained of constant and severe pain aggravated by attempts to swallow—*Case* 4 described below.

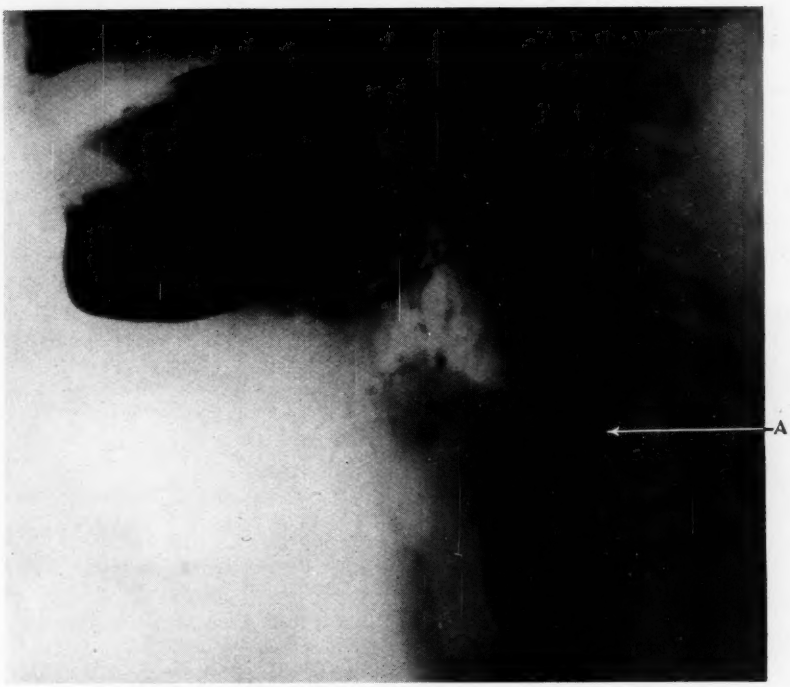


FIG. 71.—*Case* 3. A, Carcinomatous destruction of seventh cervical vertebra.

Sympathetic Nerve.—Sympathetic-nerve involvement was not observed in the present series. Abel[7] and Guisez[22] both call attention to the symptoms of such a complication—an early irritative lesion followed by paralysis with its characteristic unilateral signs (Horner's syndrome).

Thyroid Gland.—Direct spread of upper œsophageal growths into the thyroid gland does occur, and one case was noted in the present series :—

Case 4.—A. L., female, aged 54 years. Stated that she had experienced severe pain in her throat and difficulty in swallowing for the past four months, and that one month ago she had noticed a swelling of the left side of her neck. Examination showed a hard fixed enlargement of the left lobe of the thyroid gland, and paralysis of the left vocal cord. Endoscopy revealed an extensive carcinoma of the upper œsophagus involving the trachea and thyroid gland. The patient became very dyspnœic, and an emergency tracheotomy was necessary one week after her first application to hospital.

Respiratory Tract.—Involvement of the trachea, the bronchi, pleuræ, or lungs is probably the next most common complication observed during life after recurrent-nerve involvement, and it is in a large proportion of cases the immediate

cause of death. Souttar[5] has stated that 'of every 10 cases of carcinoma of the œsophagus' 9 will die from purulent bronchopneumonia and 1 from hæmorrhage. In Watson's[1] series 48 per cent of the cases coming to autopsy had died from bronchopneumonia.

Keefer[25] has reported a series of 17 cases of carcinoma of the œsophagus in which the dominant symptoms were pulmonary, and he has tabulated the causes of such respiratory complications as follows :—

1. Aspiration owing to œsophageal obstruction.
2. Non-perforating involvement of the trachea, bronchus, pleura, or lung.
3. Tracheo-bronchial stenosis due to (*a*) Direct spread of growth, or (*b*) Pressure by metastatic glands.
4. Perforation by growth into trachea, bronchus, pleura, or lung.
5. Perforation by metastatic glands into trachea, bronchus, pleura, or lung.

Such lesions are characterized by cough, hæmoptysis, and dyspnœa.

Three cases of the present series showed a non-perforating lesion of the trachea at the time of the first examination :—

Case 5.—F. C., male, aged 61 years. Complained of dysphagia of three months' duration and of coughing when lying in bed. He gave a history of syphilis thirty years previously, but his blood Wassermann reaction was negative. X-ray report: 'Marked dilatation of the upper portion of the œsophagus. The X-ray appearances point to a malignant stricture rather than the presence of a pouch.' Œsophagoscopy showed a hard, ulcerated stricture 10½ in. from the incisor teeth, which was very difficult to dilate for intubation as bleeding was very profuse. Tracheoscopy revealed a granulating mass of growth on the posterior tracheal wall situated immediately above the bifurcation (*Fig.* 72). The patient died one month later, and the post-mortem report was as follows : 'Right-sided pneumothorax—pleural cavity contained about two pints of fluid. Œsophagus—Souttar's tube in position. Œsophageal growth around the wall has ulcerated into trachea—no perforation. Numerous secondary glands. Liver normal.'

FIG. 72.—*Case* 5. Non-perforating involvement of the trachea from œsophageal carcinoma.

Case 6.—W. T. M., male, aged 59 years. Complained of difficulty in swallowing of two months' duration, and more recently of nocturnal cough. X-ray examination showed 'malignant disease of the œsophagus at level of fourth to seventh dorsal vertebræ.' The upper margin of an œsophageal neoplasm was found by œsophagoscopy at 9½ in. from the incisor teeth. Tracheoscopy revealed a similar lesion to that observed in *Case* 5.

The third case is reported above (*Case* 4).

It is of interest to note that *Cases* 5 and 6 both complained of a cough which was troublesome when in the supine position. A third patient also complained of a troublesome cough at night. He was treated with radon seeds two years ago for malignant disease of the upper third of his œsophagus and is still alive. He was examined recently and it was found that he had paralysis of his left vocal cord and a cough which interfered with his sleep at night, but was not troublesome during the day. In view of the two cases reported above, it is presumed that he, also, has a non-perforating extension to his trachea.

Nocturnal cough as a symptom of non-perforating tracheal involvement does not appear to have been described elsewhere, and it is possibly due to irritation caused by the backward pressure of the trachea on to the ulcerating mass when the patient adopts the supine position.

Souttar[26] has recorded one case in which a nodule of growth in the trachea spreading from an œsophageal neoplasm actually caused respiratory embarrassment, and one case of the present series (*Case* 4) required tracheotomy owing to tracheal stenosis due to a non-perforating involvement of the trachea by direct spread.

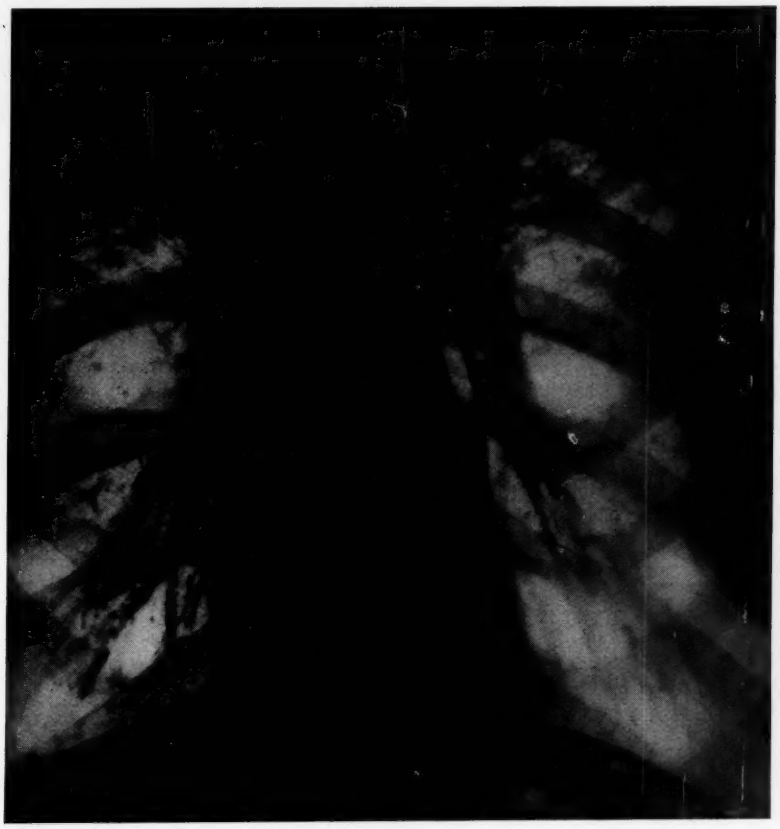


FIG. 73.—Barium paste in the bronchi after swallowing in a patient with a carcinomatous œsophago-tracheal fistula.

Perforation of the trachea or bronchi is characterized by a cough which occurs in violent paroxysms and suffocative spasms during attempts to swallow, and this is usually accompanied by fœtid expectoration. In such cases, if the patients can take barium paste, it will be seen to pass into the trachea and bronchi, partially outlining the bronchial tree (*Fig.* 73). Kerley[27] has described how he watched such a happening under the fluorescent screen in a patient with carcinoma of the middle third of the œsophagus. The barium passed into the trachea and down to the bases of the lungs, and the patient became very distressed and dyspnœic; half an hour afterwards he was quite well, and the examination was repeated some time later with similar results. No lung complications ensued, and the patient did not die until some months later, and then owing to cachexia and exhaustion.

There was only one patient in the present series who developed a tracheal fistula after diagnosis of the œsophageal lesion was made, and it was impossible to X-ray him, as the ingestion of the smallest amount of fluid caused a severe paroxysm of coughing and great distress :—

Case 7.—V. R., male, aged 62 years. Complained of dysphagia of three months' duration. X-ray and œsophagoscopy showed a malignant stricture 11 in. from the incisor teeth. This was dilated and treated with radon seeds. One month later he returned to hospital complaining of inability to swallow owing to 'choking' and of putrid purulent expectoration. He died six and a half weeks later, and autopsy showed a large perforation which admitted two fingers from the anterior wall of the œsophagus into the posterior wall of the trachea (*Fig.* 74).

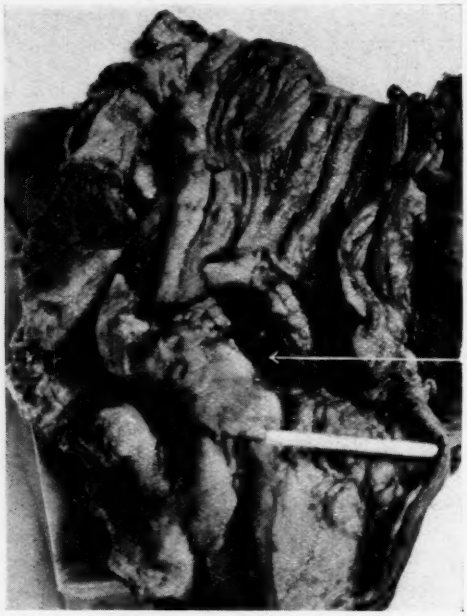


FIG. 74.—*Case* 7. A, Perforation from œsophagus into trachea.

Such a complication is an urgent indication for gastrostomy.

Guisez[28] has stated that he has observed tracheal involvement in 15 per cent of his cases, and he quotes Zenker and Ziemssen's series of 120 cases with 26 perforations into the left bronchus and 20 into the trachea. Scott-Brown[29] described 4 cases of fistula into the air-passages ; he recorded that one of these patients lived for seven months after the diagnosis of perforation was made, and Grey Turner[37] has reported the case of a man who lived for nine months with an œsophago-tracheal fistula. Watson[1] has stated that of his series of 27 post-mortem examinations 7 cases showed extension to or actual rupture into the trachea or bronchus ; and Souttar[5] has reported 3 cases with perforation into the left bronchus and 3 into the trachea itself of his series of 18 autopsies.

Involvement of the pleuræ and lungs frequently becomes manifest in the terminal phases of carcinoma of the œsophagus, and this complication is commonly observed at autopsy. When pleural pain occurs it is usually a *very* late symptom. Clayton[12] has reported 6 cases who had complained of such pain out of a series of 41 autopsies.

One patient of the present series complained of pleural pain at her first visit to hospital :—

Case 8.—S. B., female, aged 52 years. Complained of dysphagia of four months' duration and that latterly she had suffered from pain in her right chest. X rays showed irregularity of the barium shadow from the level of the seventh dorsal vertebra to the eleventh dorsal vertebra with dilatation above this region. A few days later an effusion into her right pleural cavity became evident. She was admitted to hospital, where she died three weeks later. The post-mortem examination report was as follows : 'In the lower third of the œsophagus and extending for 4 in. is a large epitheliomatous ulcer ; the growth almost blocks the lumen. Its lower extremity extends to 1 in. of the cardiac orifice. The right pleural cavity is filled with purulent fluid. Leak in the cancerous wall of the œsophagus. Mass of glands in neighbourhood of head of pancreas.'

LYMPHATIC SPREAD

The œsophagus is richly supplied with lymphatics, which are present as two networks—a submucous and a muscular. These are continuous with similar

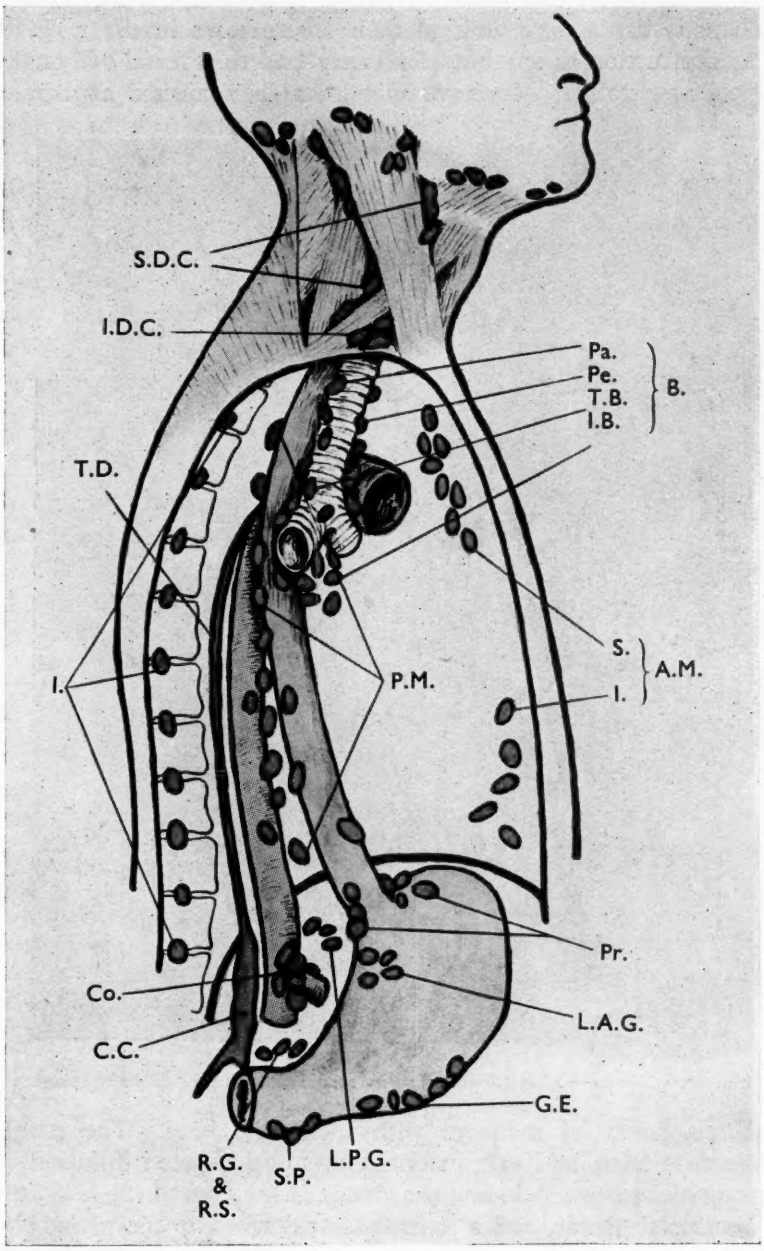


FIG. 75.—Lymphatic drainage of the œsophagus. S.D.C. and I.D.C., Superior and inferior deep cervical; Pa., Paratracheal; Pe., Pretracheal: T.B., Tracheo-bronchial; I.B., Interbronchial; B., Bronchial; S. and I.A.M., Superior and inferior anterior mediastinal; P.M., Posterior mediastinal; I., Intercostal; T.D., Thoracic duct; C.C., Cisterna chyli; Co., Coeliac: Pr., Paracardial: L.A.G., Left anterior gastric; G.E., Gastro-epiploic; L.P.G., Left posterior gastric: S.P., Subpyloric; R.G., Right gastro-epiploic; R.S., Right suprapancreatic.

lymphatic systems of the pharynx above and the stomach below. The efferents from these networks drain into the neighbouring lymphatic glands (*Fig.* 75). Those from the upper half drain into the posterior mediastinal, the deep cervical, and paratracheal glands: those from the lower half drain into the posterior mediastinal, paracardial, left anterior, and posterior gastric glands, and thence into the cœliac glands. There is hardly any limit to their anastomoses in the neck, thorax, or abdomen, as obstruction in any one gland may lead to reversal of lymph-flow and opening up of new paths. Musgrave Woodman[9] has injected experimentally the

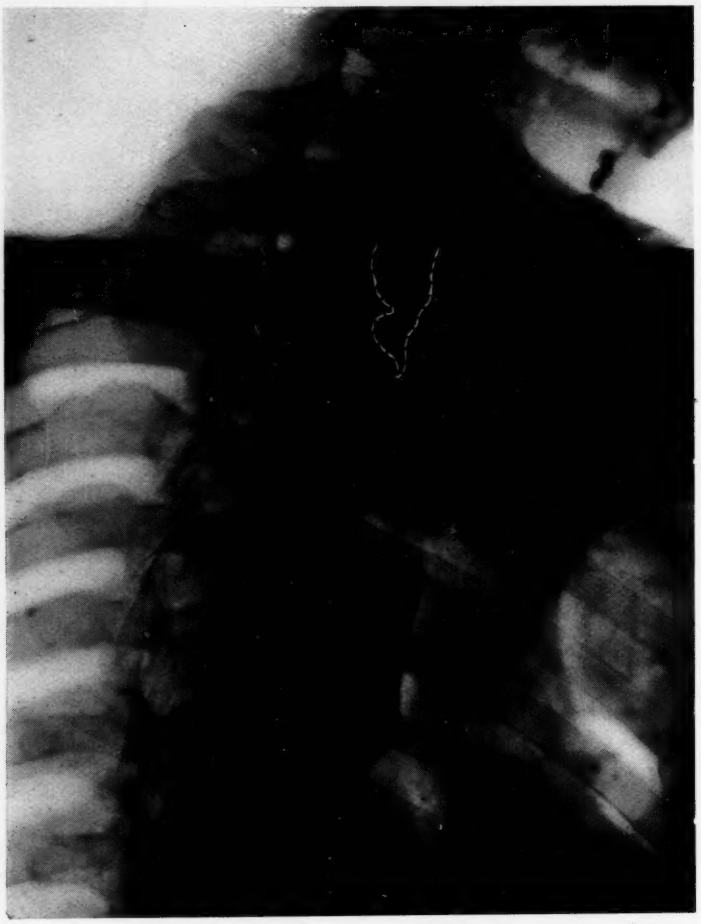


FIG. 76.—*Case* 1.—Radiogram showing two malignant strictures of the œsophagus.

œsophageal lymphatics of monkeys with methylene blue. The monkeys were killed twelve days later, and examination showed widespread diffusion of the dye into the lymphatic system, including the glands at the base of the neck, the mediastinal and intercostal glands, and a remarkable extension to the parietal pleura and the pleural surface of the diaphragm. The inference he draws from his experiments is that lymphatic spread in the early stages of carcinoma is slow and takes place with difficulty, but when once the fine reticulum has been broken through the invasion is widespread and comparatively rapid.

Permeation of the local mural lymphatics may take place, and off-shoots from these to the surface of the œsophagus may give rise to apparently multiple new growths with gaps of normal mucous membrane separating the neoplastic areas. Although this method of spread is often seen at autopsy, it is rarely encountered during life—probably because it is difficult and dangerous to pass an œsophagoscope through a malignant stricture and so diagnose a growth lower down. Guisez[30] has described two cases of multiple ulcers of the œsophagus seen during life—one case with three ulcers, and one with two. Chevalier Jackson[2] saw multiple ulcers twice only in a series of 671 cases. Souttar[31] states that these neoplastic upshoots may give rise to multiple strictures of the œsophagus, and an illustration of this complication is reproduced in *Fig.* 76. Four cases of multiple œsophageal ulcers occurred in the present series, two of which have been reported elsewhere.[32] Briefly they were as follows :—

Case 9.—G. B., female, aged 46 years. Gave a history of four months' dysphagia. X rays, œsophagoscopy, and biopsy showed an annular carcinomatous ulcer 10 in. from the incisor teeth. Radon seeds were inserted. Three months later she returned with a malignant stricture situated at 12 in. from the incisor teeth, the original ulcer being healed as a fibrous stricture.

Case 10.—J. O., male, aged 65 years. Complained of dysphagia of nine months' duration. Œsophagoscopy showed ulcers situated at 7½ in and 11½ in. from the incisor teeth. Snippings from both ulcers were reported to be epitheliomatous by Professor Beattie.

Case 1.—W. A. B., male, aged 60 years. Came to hospital suffering from bilateral recurrent nerve paralysis, and his symptoms have been described above. He was very emaciated; and X-ray examination by Dr. Whitaker demonstrated two strictures of the œsophagus (*Fig.* 76). Œsophagoscopy showed a malignant stricture at the entrance of the œsophagus and another situated at 13 in. from the incisor teeth. Biopsy proved both growths to be epitheliomatous.

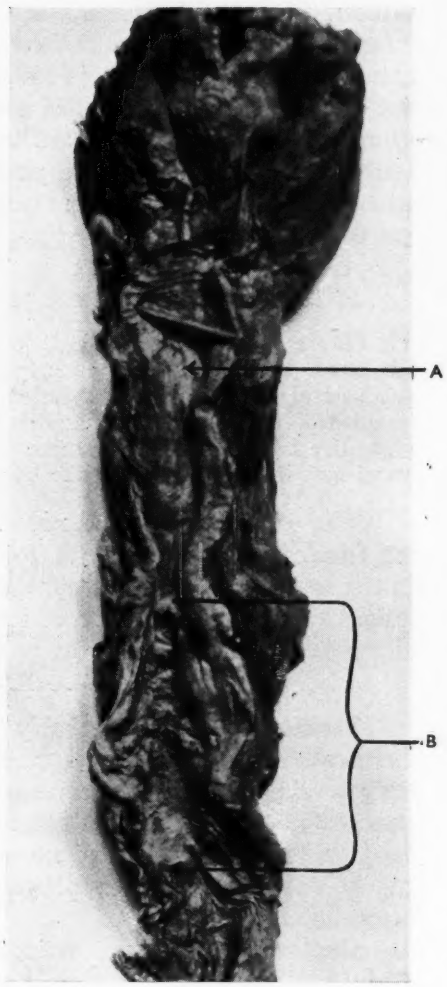


FIG. 77.—*Case* 11. A, Superior ulcer; B, Inferior ulcer.

Case 11.—R. W., male, aged 62 years. Complained of dysphagia of eight weeks' duration. X rays showed a 'stricture, probably malignant, of upper third of the œsophagus.' Œsophagoscopy demonstrated a submucous non-ulcerated growth 8¼ in. from the incisors and a large annular malignant stricture at the lower end of the œsophagus. This patient died four months later and autopsy showed a small growth of the upper third of the œsophagus just beginning to ulcerate, and a large ulcerated growth of the lower third (*Fig.* 77). Many of the mediastinal structures were involved by extra-œsophageal growth—pleuræ, pericardium, etc.—and there were numerous glandular secondaries in the cœliac lymphatic group.

Multiple strictures may also be caused by pressure of enlarged metastatic glands. Keefer[25] has reported such a case. Œsophageal obstruction due to a secondary glandular mass occurred in one case of the present series (*Case* 13) which is reported later. This was observed only at autopsy and was not diagnosed during life.

There can be no doubt that lymphatic glandular deposits and lymphatic extension to more distant organs is commonly seen at autopsy—yet the question of the frequency of secondary lymphatic involvement is unsettled and is fully discussed by Abel,[7] Souttar,[5] and Watson.[1] Only 13 cases of the present series have come to autopsy, and of these 10 showed macroscopic lymphatic-gland involvement in the thorax and/or in the abdomen. The presence of metastatic glands is not often recognized during life, as the patients usually succumb before recognizable symptoms attributable to glandular enlargement become manifest. Souttar[26] has stated that masses of enlarged glands may become manifest in the neck or epigastrium of patients suffering from carcinoma of the œsophagus, and Chevalier Jackson[2] recorded that out of a series of 671 cases in 16 the œsophageal lesion gave rise to no symptoms and was only discovered in seeking for a cause of cervical adenopathy.

Five cases occurred in the present series :—

Case 12.—F. E., female, aged 53 years. Referred from Mr. W. R. Williams's Clinic for investigation, with enlarged palpable glands in her left supraclavicular triangle. The enlargement was of six weeks' duration, and the patient made no complaint of dysphagia until questioned. X rays showed some delay of opaque paste at the upper end of the œsophagus, suggestive of a post-cricoid carcinoma. Œsophagoscopy revealed a malignant ulcer of the œsophagus immediately below the cricoid.

The remaining four cases have already been mentioned and were males suffering from right recurrent-nerve palsy. In all four cases the growths were situated in the lower third of the œsophagus, and it is reasonable to assume that the nerve lesions were due to pressure or involvement by glandular metastases.

BLOOD-STREAM SPREAD

Venous spread is probably rare. Abel[4] has recounted the findings of Leichenstein, who observed a case in which direct extension took place along the azygos vein to the superior vena cava and thence to the right auricle and right ventricle; and Souttar[5] has recorded the post-mortem findings in a male patient aged 62 years who exhibited no signs of carcinoma of the œsophagus during life, but at autopsy a large œsophageal carcinoma was found with metastases in the cerebellum.

One case of the present series is probably an example of blood-borne spread :—

Case 13.—M. A. B., female, aged 54 years. Stated that she had pain in her chest for two months when swallowing. X rays, œsophagoscopy, and biopsy showed a malignant ulcer of her œsophagus at 9 in. from the incisor teeth. This was treated with radium. A month later she complained of a lump on her right hip, and she entered another hospital, where she died seven months later. I am indebted to Professor T. B. Davie for the following post-mortem report: 'Thorax: large secondary mass growing in eleventh rib right up to vertebral column—pleural surface not involved. Lungs: one secondary mass near left apex involving lung tissue but possibly only secondarily from a gland metastasis. This mass is adherent to the outside of the œsophagus and is visible from within the œsophageal lumen as a protuberance just above the tracheal bifurcation. Œsophagus: no present ulceration anywhere. At level of tracheal bifurcation the lumen is slightly obstructed by protuberance inward over adherent gland mass—no definite scar within the œsophagus. Abdomen: one gland mass

on lesser curvature just below diaphragm. No growth in stomach. Liver, kidneys, and spleen: all normal. Adrenals: left involved in malignant metastases. Bones: large metastasis on surface of right iliac bone eroding bone substance.'

SPREAD BY IMPLANTATION

Spread by implantation is rare, and when this does occur in the œsophagus itself it is impossible to state definitely without a careful microscopical examination that the secondary lesion is not due to spread by the deep lymphatics as described above.

Two cases of undoubted implantation spread occurred in the present series, and a third case is described which appeared to be an example of endo-œsophageal spread by this method. One of the former cases has been recorded elsewhere.[32]

FIG. 78.—*Case* 15. Multiple oral neoplastic ulcers associated with carcinoma of the œsophagus.

Case 14.—R. K., male, aged 62 years. Was treated at the Royal Southern Hospital with radium in November, 1932, for a carcinomatous stricture of the œsophagus at 13½ in. from the incisor teeth. In February, 1933, radium was applied to a carcinoma of the left side of his tongue by Mr. R. Kennon at the Liverpool Royal Infirmary. He died in August, 1933, with numerous metastases from the lingual and œsophageal primaries.

The second case was seen more recently by the courtesy of Mr. W. R. Williams:—

Case 15.—H. H., male, aged 62 years. Stated that four months previously he first noticed some difficulty when swallowing. Two months later he had suffered from bronchitis and pleurisy, since when swallowing had been more difficult, and he could only take thick liquids at the time of his admission to hospital. A lump had appeared in the left side of his neck two months ago and on the right side six weeks ago—neither of which was painful. His throat had been sore for the past three weeks. Examination showed a large neoplastic ulcer involving the right anterior faucial pillar, hard palate, and buccal surface of the right cheek;

and a smaller ulcer of the buccal mucosa situated at the region of the junction of the ascending and horizontal rami of the mandible (*Fig.* 78). There were hard fixed masses of inoperable glands situated in both anterior triangles. X-ray examination of the œsophagus disclosed a 'hold-up of barium at level of seventh dorsal vertebra due to malignant stricture of œsophagus — ? specific.' Blood Wassermann reaction was negative. Œsophagoscopy revealed an extensive annular carcinomatous stricture of the œsophagus 13 in. from the incisor teeth. Snippings from both faucial ulcers and from the œsophageal growth were reported by Dr. R. Mole to be epitheliomatous. A Souttar's tube was inserted through the stricture, but the patient died two months later. No autopsy was obtained.

The third case exhibited what appeared to be an implantation growth in the œsophagus itself—this was a presumptive diagnosis only, as it was impossible to prove that the spread was not due to lymphatic permeation :—

Case 16.—W. P., male, aged 62 years. Complained of a 'feeling of something in his throat' when swallowing of four weeks' duration. X rays demonstrated a partial hold-up immediately below the cricoid. Œsophagoscopy showed a proliferating ulcer of the anterior wall of the œsophagus 7½ in. from the incisor teeth. Immediately opposite to this on the posterior wall was a small solitary nodule of growth (*Fig.* 79). The mucous membrane separating the growths was normal. There appeared to be no infiltration of these areas, and the nodule suggested a surface infection —a true implantation growth.

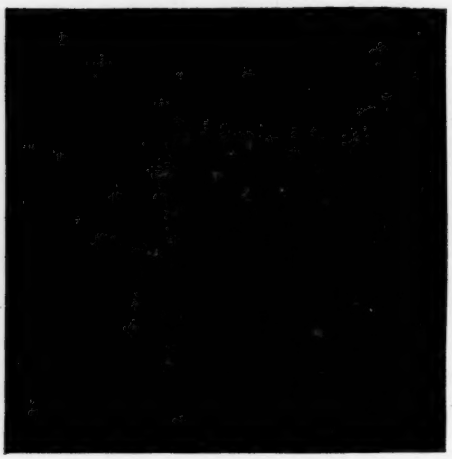


FIG. 79.—*Case* 16. Multiple growths of œsophagus.

The two former cases are very unusual, and I can find only one reference to proved implantation secondary growths from an œsophageal carcinoma. Hoche[33] described a case in which an epithelioma arose on a gastric ulcer in a patient suffering from carcinoma of the œsophagus.

CACHEXIA

Five patients of the present series arrived at hospital in an advanced state of starvation and exhaustion. All were considered to be unfit for palliative treatment, even by dilatation or intubation. Four cases were admitted forthwith and treated with sedatives and proctoclysis. The fifth case was sent home to await admission, but he was found dead in bed on the following day.

SURGICAL REMOVAL

Only one patient of the present series of 100 cases was considered suitable for an attempt at excision, but operation proved the growth to be too extensive for removal. The case was examined by the kindness of Mr. C. A. Wells, who subsequently operated :—

Case 17.—P. H., male, aged 44 years, gave a history of four months' dysphagia. X-ray report : 'Hold-up at extreme upper end of œsophagus.' Œsophagoscopy showed the upper edge of a proliferating neoplasm 1 in. below the cricoid. The visible growth appeared to be situated on the right lateral wall and suggested a comparatively localized lesion. The

œsophagoscope was not passed through the neoplastic area. A gastrostomy was performed by Mr. Wells, who later exposed the upper œsophagus through a right cervical incision, intending to use a Trotter's skin-flap. The growth was found to be very extensive, passing down for some inches into the mediastinum and adhering to and infiltrating all the neighbouring prevertebral structures. The operation was abandoned and two days later a leak from the œsophagus into the neck wound became manifest. The patient died within a short period. No autopsy was performed.

COMMENTARY

The case histories of 100 cases of carcinoma of the œsophagus have been examined in order to ascertain the reason for the non-performance of radical surgical treatment.

Of this series in one case excision of the tumour was contemplated, but operation proved the lesion to be too extensive for removal. Severe cachexia and exhaustion was shown by five patients on their arrival at hospital so that simple alleviation only of their suffering until death was possible. In 65 cases X rays and œsophagoscopy revealed so extensive a local lesion that palliative treatment alone was considered to be feasible. In 29 cases evidence of spread of the growth was present at or about the time of the first application for treatment.

This latter figure is a surprisingly high one, and the findings bear witness to the tragedy of late diagnosis. The loss in this manner of valuable time is due largely to lack of appreciation by the general public of the gravity of any abnormality of the act of swallowing, and of the necessity at the first occurrence of such complaint for a complete clinical, radiographic, œsophagoscopic, and bronchoscopic investigation. This will certainly involve the performance of many examinations with negative results, but it is the only routine by which at present the early case can be discovered. No investigation of a suspected case of œsophageal neoplasm is complete without direct inspection of the trachea and larger bronchi, and this procedure should be carried out at whatever level the œsophageal lesion is situated, as not only may the primary growth involve any portion of the lower respiratory tract, but such a complication may be due to invasion from metastases.

The course of carcinoma of the œsophagus can be divided clinically into three phases :—

1. The latent period—which is symptomless and of unknown duration.

2. The 'symptom' period—i.e., the time lost between the incidence of the earliest symptoms and the date of a definite diagnosis. This period averages 5·7 months.

3. The 'manifest' period, i.e., the survival time of the patient from the date of diagnosis until death. This period averages 5·14 months.

Chevalier Jackson,[2] who has tabulated the early symptoms of carcinoma of the œsophagus, is of the opinion—with which all those who are interested in the subject will agree—that in medical education the importance of the so-called late symptoms should be minimized, whereas the description of the early symptoms, which has not yet found its way into text-books, should be emphasized. He has further stated[34] that 'not until every patient who complains of cervical, retro-sternal or epigastric abnormal sensation is regarded *not* as neurotic but as possibly cancerous, will the surgeon have a chance.'

The results of the above analysis are not submitted as a discouragement to

attempts at radical surgical methods. That such methods are firmly established on a secure technique is evidenced by the brilliant achievements of British and Continental surgeons—of the former Professor Grey Turner[35] and Mr. King[36] recently have reported successful cases of removal of the entire thoracic œsophagus. The findings, however, would indicate that if diagnosis was made at the onset of the 'symptom' period, then Abel's contention that at least one case out of every four could be cured by radical surgical methods might be tenable.

In addition to the acknowledgements made in the text, I am indebted to my colleagues at the Royal Southern Hospital for permission to examine and treat their cases, and particularly are my thanks due to Drs. J. H. Mather, R. E. Roberts, P. Whitaker, and A. E. Connolly for numerous expert X-ray examinations without which it would have been impossible to record these notes.

REFERENCES

[1] WATSON, W. L., *Surg. Gynecol. and Obst.*, 1933, lvi, 884.
[2] JACKSON, CHEVALIER, *Amer. Jour. Med. Sci.*, 1925, clxix, 625.
[3] JACKSON, CHEVALIER, *Bronchoscopy and Œsophagoscopy*, 1927, 296.
[4] ABEL, A. L., *Brit. Jour. Surg.*, 1926, xiv, 131.
[5] SOUTTAR, H. S., *Ibid.*, 1927, xv, 71.
[6] BRODERS, A. C., and VINSON, P. P., *Arch. Otol.*, 1928, viii, 79.
[7] ABEL, A. L., *Œsophageal Obstruction*, 1927, 168.
[8] WRIGHT, A. J., *Brit. Jour. Surg.*, 1927, xv, 71.
[9] WOODMAN, E. MUSGRAVE, *Brit. Med. Jour.*, 1931, ii, 290.
[10] VINSON, P. P., *Proc. Staff Meeting Mayo Clinic*, 1931, June 14.
[11] ROBERTS, O. W., *Brit. Jour. Surg.*, 1930, xvii, 683.
[12] CLAYTON, E. S., *Surg. Gynecol. and Obst.*, 1928, xlvi, 52.
[13] HELSLEY, F. G., *Ann. of Surg.*, 1928, lxxvii, 272.
[14] EWING, J., *Neoplastic Disease*, 1928, 904.
[15] ILLINGWORTH, C. F. W., and DICK, B. M., *Textbook of Pathology*, 1932, 409.
[16] LOWE, E. CRONIN, Personal communication.
[17] Quoted by Watson.[1]
[18] Quoted by Abel.[7]
[19] CLEMINSON, F. J., and MONKHOUSE, J. P., *Jour. Laryngol. and Otol.*, 1934, xlix, 313.
[20] GRAHAM, E. A., and BALLON, H. C., *Ann. of Otol. Rhinol. and Laryngol.*, 1931, xl, 896.
[21] SEMON, SIR F., *Arch. Otolaryngol.*, 1881, ii, 197.
[22] GUISEZ, J., *Bull et Mém. Soc. de Chir.*, 1930, xxii, 751.
[23] GUISEZ, J., *Rétrécissements de l'Œsophage*, 1923.
[24] NEW, G. B., and CHIDLEY, J. H., *Arch. Otolaryngol.*, 1932, xvi, 143.
[25] KEEFER, C. S., *Ann. of Internal Med.*, 1934, viii, 72.
[26] SOUTTAR, H. S., *Brit. Med. Jour.*, 1934, ii, 798.
[27] KERLEY, P., *Recent Advances in Radiology*, 1931, 195.
[28] GUISEZ, J., *Rétrécissements de l'Œsophage*, 1923, 139.
[29] SCOTT-BROWN, W. G., *Lancet*, 1934, ii, 544.
[30] GUISEZ, J., *Rétrécissements de l'Œsophage*, 1923, 154.
[31] SOUTTAR, H. S., *Choyce's System of Surgery*, 1932, 184.
[32] MCGIBBON, J. E. G., *Lancet*, 1933, ii, 806.
[33] HOCHE, L., *Presse méd.*, 1901, i, 67.
[34] JACKSON, CHEVALIER, *Ann. Otol. Rhinol. and Laryngol.*, 1931, xl, 889.
[35] TURNER, G. GREY, *Proc. Roy. Soc. Med.*, 1934, xxvii, 355.
[36] KING, E. S. J., *Brit. Jour. Surg.*, 1936, xxiii, 521.
[37] TURNER, G. GREY, *Lancet*, 1936, i, 68.

INTRAVENOUS INJECTION OF HYPERTONIC SODIUM CHLORIDE SOLUTION IN THE TREATMENT OF SOME CONDITIONS OF LOW BLOOD-PRESSURE

By JOAN B. WALKER, Leicester

Hypertonic sodium chloride solution has been used intravenously for several different purposes. It has been given to reduce an increased intracranial pressure from œdema or hæmorrhage ;[1] to promote intestinal motility in cases of paralytic ileus ;[2] and in cholera[3] it has been given in order to attract water to the blood, which is viscous owing to extreme dehydration of the body. It has not been in general use for the purpose of raising a lowered blood-pressure, although it has been suggested by Graham[4] that a pint of 1·2 per cent sodium chloride should be given to raise the blood-pressure in diabetic coma, as in this condition the tissues lack water and chlorine, and sodium chloride also helps to neutralize acetone bodies. Other workers have put forward objections to the use of hypertonic solutions in the treatment of shock. Bayliss,[5] in his Oliver Sharpey Lectures of 1918, reviewed the physiological and therapeutic action of various solutions that could be employed by intravenous injection for the treatment of wound shock. He came to the conclusion that a hypertonic solution of sodium chloride was inadvisable as he found the effect transitory and he had noticed unpleasant symptoms of gasping respiration and slowing of the heart. He stated that he believed it undesirable to add to the saline content of the organism, which is an interesting observation when considering the recent treatment of Addison's disease, with its associated low blood-pressure, by sodium chloride.[6, 7] If it is undesirable to add to, it seems essential to maintain, the balance of circulating sodium chloride, both on account of its chemical and of its physical properties.

MacFee and Baldridge,[8] also when considering the infusion treatment of shock, state that they ' see small logic in the use of hypertonic solutions ', because of the temporary hydræmia, rapid excretion by the kidneys, and finally a loss rather than gain of fluid.

Cushing and Foley[9] find that the raised blood-pressure may be an objection to the use of hypertonic saline intravenously for reducing intracranial tension.

With due regard for the objections mentioned above, the present series of observations were made on 53 cases. In 49 of these hypertonic saline was injected intravenously with the object of raising a lowered blood-pressure before, during, or after operation. Most cases received 40 c.c. of a 30 per cent solution (12 grm.) of sodium chloride. Blood-pressure records were kept which show the rapidity and duration of the reaction.

The two cases which suggested the possibility of using small quantities of strongly hypertonic salt solution for its rapid stimulating effect on the circulation happened to occur on the same day. In the first case 30 c.c. of 30 per cent sodium chloride solution were injected intravenously, with the aim of reducing intracranial

tension to close the dura following an exploratory operation for cerebral tumour. A record had been kept for over two hours and the systolic blood-pressure had been between 140 and 130 mm. Hg. Within five minutes of the injection it rose from 130 to 165, but the effect was transitory.

Fig. 80 shows the record of the second case—that of a woman aged 60. Spinal anæsthesia was administered for laparotomy. Paralytic ileus due to pneumococcal peritonitis was subsequently found. There was severe initial shock; the systolic blood-pressure fell from 140 to 70 mm. Hg: 40 c.c. of 30 per cent NaCl solution were injected intravenously. This time, with the morning's case in mind, it was hoped that there would be the same quick rise in blood-pressure; also there was

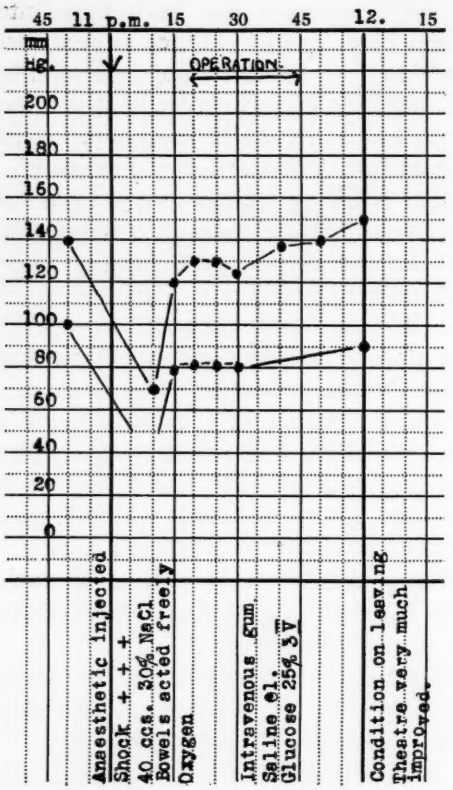


FIG. 80.—*Case* 2. Female, aged 60. Pneumococcal peritonitis.

the idea of possibly replacing the chlorides in the blood lost by her repeated vomiting, and of promoting peristalsis. It was to be followed by a pint of gum saline and glucose. Five minutes after the injection the blood-pressure rose from a systolic reading of 70 to 120. A copious evacuation of the bowels occurred, the operation was performed, and the patient returned to bed in an improved condition.

The improvement in this case was sufficiently dramatic, and so much more rapid than any obtained by the usual cardiac stimulants that it seemed reasonable to try the effect of similar injections in other cases for the specific purpose of raising the blood-pressure. Cases of spinal anæsthesia with a sudden initial fall of blood-pressure before the operation had begun and before there was any actual blood lost

outside the body were thought to be particularly suitable. It was with regard to the problem of making spinal anæsthesia as safe for the patient as it makes operating easy for the surgeon that this investigation was undertaken, and with special reference to the use of spinal anæsthesia for acute intestinal obstruction. In these cases it is of great value, but it may be dangerous for the patient who is already very ill. The advantages are that it ensures absolute muscular relaxation, which can otherwise only be obtained by deep inhalation anæsthesia of toxic substances undesirable for a toxic patient. It is safer for the patient to be conscious and able to cough and so avoid inhaling regurgitated material. The distension of the intestines is reduced and they are not extruded from the wound with each respiration. Paralysis of the sympathetics by spinal anæsthesia may cause an evacuation of the bowels as soon as the obstruction is removed. This is desirable in preventing further absorption of toxins from the fluid contents of the intestines. Breathing is quiet with spinal anæsthesia, since the intercostal nerves are blocked to a definite level, and there is no straining when the peritoneum is closed. The serious disadvantage in using spinal anæsthesia is the considerable fall in blood-pressure coincident with the onset of anæsthesia. In a case of intestinal obstruction the patient is already in a condition of shock with dehydration and loss of chlorides from the blood by vomiting, and poisoned by the absorption of toxins from the intestinal contents. He is in no condition to stand any further lowering of his blood-pressure.

THE TYPE OF SHOCK IN SPINAL ANÆSTHESIA

In 300 cases operated upon under spinal anæsthesia, in 157 (32·3 per cent) there was an initial fall in blood-pressure before the operation began in spite of some stimulating drug having been injected prophylactically. Even in an averagely healthy patient who, for example, is having an interim appendicectomy performed, there is generally a tendency for the blood-pressure to be lowered for ten to fifteen minutes after the intrathecal injection of the anæsthetic solution. This appears to bear no relation to the drug used nor to its specific gravity, but depends upon the level of anæsthesia and muscular paralysis produced. In the group of 300 cases mentioned above, there was little or no anæsthesia produced in 42 cases (14 per cent). In only 2 of these was there a fall in blood-pressure before the operation began. The sudden hypotension may cause pallor, sweating, nausea, retching or vomiting, with anxiety, restlessness, or delirium. When this condition, described by Quarella[10] as ' the storm ', is due to the anæsthesia alone spontaneous recovery may be expected to take place in twenty-five to thirty minutes. For this reason Sebrechts[11] states that it is dangerous to begin to operate within thirty-five minutes of the intrathecal injection. Howard Jones[12] explained the collapse by the absorption of the anæsthetic drug into the general circulation, but absorption from the cerebrospinal fluid to the blood is slow. In the cases of failed spinal anæsthesia where, although the usual quantity of anæsthetic has been injected with the usual technique, little or no anæsthesia or paralysis of muscles occurs, there is the same opportunity for absorption from the cerebrospinal fluid, but no fall in blood-pressure is recorded. The possibility of puncturing a vein on performing lumbar puncture, and injecting directly into it, could only occur with faulty technique, by not allowing a flow of clear cerebrospinal fluid before making the injection. This explanation of the collapse is not the most probable one.

The condition of shock coincides with the rapid onset of anæsthesia and flaccid paralysis of more than half of the muscles of the body. The two factors which appear to determine the distribution of the volume of circulating blood at this time are, firstly, the extensive flaccid muscular paralysis, and, secondly, paralysis of the sympathetics. Blood may stagnate in the vessels of the affected skeletal muscles, and in the dilated vessels of the splanchnic area, with exudation of plasma into the tissue spaces. The volume of circulating blood is reduced and is more concentrated. There is a natural tendency to restore the circulating blood-volume by drawing on the fluid reserves of the body and the recovery phase sets in.

In experimental shock Blalock[13] found that there was accumulation of blood with local loss of plasma in the traumatized areas on comparing the weights of the crushed hind limb of a dog with its normal fellow. He found that trauma never reduced the blood-pressure to a shock level without causing the loss of enough blood and plasma into the traumatized area to account for this fall. This work was confirmed by Holt and Macdonald.[14] Their experimental findings seem to correspond with the apparent distribution of blood at the onset of spinal anæsthesia, although at present absolute proof is lacking. A small practical point supporting this view is mentioned by Hughes,[15] who finds that by bandaging the lower limbs striking falls in blood-pressure are prevented. The part played by the adrenal cortex and medulla in the production of this condition of shock can only be suggested by analogy with the circulatory collapse and hypotension of Addison's disease, which it resembles.

METHODS USED IN THE PREVENTION AND TREATMENT OF THE FALL IN BLOOD-PRESSURE IN SPINAL ANÆSTHESIA

Blood-pressure may be raised by: (1) Increasing the force of the heart's contraction; (2) Increasing the peripheral resistance by vasoconstriction; (3) Increasing the depth of respiration; (4) Increasing the volume of the blood circulating.

Prophylactic injection of a pressor substance is the method commonly used to prevent the initial fall in blood-pressure. Some of the proprietary preparations of novocain contain adrenalin or strychnine, so that it is all given intrathecally. This seems to be of doubtful value because of the slow absorption of drugs from the cerebrospinal fluid into the general circulation. Ephedrine has been given alone or combined with a local anæsthetic solution for injection subcutaneously and intramuscularly at the site of the proposed lumbar puncture. It has been found to have a better stimulating effect on the circulation in a dose of 1½ gr. in preference to two or more smaller doses. It should be given at the same time as the spinal anæsthetic, or before, so that its maximum effect coincides with the onset of anæsthesia. Given later it frequently fails to raise a lowered blood-pressure, and the pressor effect is less with repeated injections. Racedrin (Bayer), racemic ephedrine, appears to be more reliable in its action.

Adrenalin and pituitrin are too powerful and too transient in their action to be of routine use, but may occasionally be of use in a sudden emergency. Coramine (Clayton Aniline Company) has been found to be a powerful respiratory stimulant without any following circulatory depression,[16] but it should be given in full doses of 3 to 4 or 5 c.c. intramuscularly or intravenously where there is severe collapse.

Icoral (Bayer), a solution of two synthetic bases—one like lobeline with a stimulating action on the respiratory centre, the other like ephedrine—stimulates the circulation but is more powerful, and the action takes place in five or ten minutes and lasts longer.

These methods of raising the blood-pressure by means of possibly some slight stimulation of the cardiac muscle, but chiefly by constricting the arterioles and raising the peripheral resistance, may be best employed prophylactically. If there is constriction of the arterioles at the onset of anæsthesia, there is less opportunity for the blood to stagnate in the paralysed lower limbs, but the spinal anæsthetic paralyses the sympathetics, antagonizing the action of the drugs injected unless they are working very strongly first.

An objection to the use of vasoconstrictors for raising the blood-pressure when shock has occurred is that the oxygen supply to the tissues is diminished by virtue of the constriction, and this at a time when more oxygen is required.

Increased depth of respiration raises the blood-pressure firstly by the increased action of the diaphragm. The suction of the thoracic contents aids the return of venous blood to the heart; a greater volume is pumped with each beat and the heart's output is increased. Secondly, there is increased oxygenation of all the tissues, including the heart muscle by the coronary arteries, and the adrenals, as pointed out by Flint[17] in his article on the use of carbon dioxide for this purpose. Some of the drugs discussed previously owe their action on the blood-pressure in part to their action on the respiratory centre in the medulla.

The continuous administration of varying percentages of carbon dioxide and oxygen increases the depth of respiration by direct action on the respiratory centre, and will raise the blood-pressure some 20 to 30 mm. Hg. This will not be sufficient to combat severe shock with very low readings.

Pure oxygen inhalation produces a rise in systolic blood-pressure without increased depth of respiration. Improved oxygenation of all the tissues will account for this.

Nitrous oxide and oxygen anæsthesia has also been found beneficial in raising the blood-pressure, owing to several combined factors. Nitrous oxide itself in small quantities has a stimulating effect; oxygen is then supplied with it and acts in the same way; a re-breathing bag is usually employed for administration, therefore a certain percentage of carbon dioxide is present and exerts its similar effect. Lastly, the fact that the patient is now completely unconscious means that there can be no further psychic shock.

The posture of the patient may be mentioned at this point, as it may affect the respiratory movements. A great deal of attention has been paid in the past to the position of the patient while administering and maintaining spinal anæsthesia. A certain amount of care and common sense is required on account of the relative density of the fluids used and the action of gravity affecting the spread of anæsthetic fluid. After about twenty minutes the anæsthesia appears to become fixed and the patient may be moved into the position most convenient for the surgeon. It is customary to treat shocked patients by blocking the foot of the bed, or by using the Trendelenburg position on the operating-table to encourage the circulation to the brain and vital centres. A tilt of 5° or 10° usually helps the patient, but a steeper incline, although strongly recommended by some authorities, may have the opposite effect to that desired. The action of the diaphragm may be impeded by the weight

of the abdominal contents, and this is particularly true with obese patients whose hearts are loaded with fat. The venous return to the heart is not so good, and sometimes the lower blood-pressure records are met when the patient is steeply tilted.

The question of the volume of fluid in the circulation being maintained at a normal level is of great importance. When the fluid is actually lost from the body by hæmorrhage, it is best restored by transfusion of whole blood. When this cannot be obtained, isotonic saline solution with gum acacia, the colloidal properties[5] of which prevent the passage of the solution out of the circulation, may be given intravenously instead, but it lacks hæmoglobin and antibodies of value in the recovery phase. These are the measures preferred at the end of a long operation where there has been blood-loss.

In cases of intestinal obstruction fluid and salt are lost from the body by vomiting. There is thus a condition of dehydration with exudation of plasma into the tissue spaces leaving behind a diminished volume of blood in the circulation with a high percentage of hæmoglobin, a raised blood-urea, and lowered chloride content. Before operation the condition is improved and the blood-pressure raised to some degree by the infusion of one or more pints of isotonic saline. Of interest in connection with the rate at which normal saline leaves the circulation is the experimental work of Boycott,[18] and also of Smith and Mendel.[19] They injected Ringer's solution into the jugular vein of rabbits without bleeding them. The dilution of the blood was tested at five-minute intervals and showed that it had returned to the normal volume in twenty-five minutes; 67 per cent of the fluid escaped within five minutes, and there was no œdema or pleural effusion. Engels[20] found that the muscles, which are approximately 40 per cent of the total body weight, contained 60 per cent of the fluid after intravenous injection. He was using dogs which had been deprived of water and food for four days. Whether the solution is used with gum acacia or not, when the spinal anæsthetic is administered the blood-pressure is likely to become lowered as it takes effect, but probably not to the same extent as without a preliminary intravenous infusion. Other factors play their part and must be considered—namely, the age and nutrition of the patient, the length of time the obstruction has been present, and its position.

For the reason that under spinal anæsthesia there appears to be a temporary loss of fluid from the circulation into the paralysed muscles and tissue spaces and not an actual loss of fluid from the body, it seems reasonable to use a small quantity of strongly hypertonic sodium chloride solution which as an electrolyte exerts a high osmotic pressure and rapidly restores fluid to the circulation and tides over a period of crisis. No extra fluid volume is used which can embarrass the heart's action, and no vasoconstriction occurs to diminish the oxygen supply to the tissues.

On injecting 40 c.c. of 30 per cent sodium chloride solution into a vein in a patient suffering from the initial collapse following spinal anæsthesia, there is usually a dramatic response in five minutes which is more rapid than any found with the circulatory stimulant drugs mentioned above. The patient, instead of being restless, anxious, sweating, retching, and cyanosed, becomes pink, takes a deep breath, and if conscious will be comfortable and tell you he feels better. The pulse, from being impalpable or very weak, increases in volume and becomes slower in rate. This can be checked by blood-pressure readings taken at two-and-a-half-minute intervals after the injection. The systolic pressure rises rapidly and there is a greater

pulse-pressure. The clinical picture resembles very closely the response of a patient with Addison's disease in crisis to treatment with intravenous sodium chloride and cortical adrenal extract, the difference being that in the cases under consideration the adrenal glands are presumably not permanently diseased, but may be temporarily disturbed.

SOME PHYSIOLOGICAL EFFECTS OF HYPERTONIC SODIUM CHLORIDE

When hypertonic sodium chloride solution is injected into a vein it exerts a high osmotic pressure. Water is drawn from the tissues into the circulation during the period when the plasma is hypertonic.[21] There is a transitory increase in the plasma volume and dilution of the plasma proteins. The colloidal osmotic pressure is reduced, diuresis results, and the blood-volume may then fall below normal by the water lost in the urine. The diuresis itself may be of use in helping the body to get rid of acid products and toxic substances produced while there is diminished activity of all tissues during the period of shock when there is a reduced oxygen supply and consequent accumulation of waste material.

Hypothetically sodium chloride may combine with toxin to form a non-toxic product, with a resulting alkalosis. Richet, quoted by Barber and Oriel,[22] has shown that experimental anaphylactic shock could be prevented by the previous injection of sodium chloride. It has, on the other hand, been shown experimentally that the flow of urine which follows the intravenous injection of hypertonic saline is checked if a condition of acidosis is produced. The uræmia syndrome[23] occurs through a disturbance in the balance of acid base and mineral salt. No cases of uræmia were noted in the present series of cases, but blood-urea estimations were not made. Those deaths occurring soon after operation were in cases which were almost hopeless operative risks and could not be attributed to hypertonic sodium chloride.

There is a change in colour on increasing the sodium chloride[21] in plasma. Varying carbon dioxide tension in the blood alters the distribution of chlorine as well as HCO_3 between cells and plasma. If the carbon dioxide tension is raised, more H_2CO_3 is formed, combining with part of the base previously bound by hæmoglobin, forming potassium bicarbonate in place of potassium hæmoglobinate. The electrolytic action of NaCl may cause a reversal of the reduced hæmoglobin to normal oxyhæmoglobin. Clinically the rapid change from pallor and cyanosis to a pink colour is seen immediately in the face and hands when extra sodium chloride has been given. The explanation may not be entirely chemical, but in part due to the raised blood-pressure causing better oxygenation of the blood in the lungs.

A complaint of thirst is made by many patients soon after the injection of hypertonic saline. The dryness of the throat and defective secretion of saliva may be explained by the osmotic mechanism of the secretory cells by which water is passed from the blood to the ducts functioning under greater difficulties because more work is required to remove water from a concentrated solution than from a dilute.

Sudden severe headache may occur immediately after the intravenous injection has been made, and must be attributed not so much to the sudden lowering of intracranial tension as to the sudden increased blood-supply to the brain before the change in flow of cerebrospinal fluid occurs.[24] It is a very temporary headache and usually has passed off within ten minutes and does not recur.

Increased peristalsis and evacuation of the bowels occurred in a relatively low percentage of cases, but the small intestine may empty itself into the large bowel without complete evacuation. Necrosis of the skin occurred in 3 cases where a small quantity of the hypertonic saline was injected subcutaneously by an error of technique. The skin becomes dehydrated and changes colour from normal to dead white to brown within a few minutes. Later a slough separates. Thrombosis of a vein occurred in two cases where small veins were used and the injection was made with difficulty and slowness. No thrombosis occurred where large-sized veins were used such as those at the bend of the elbow.

CASE RECORDS

Of 53 cases where the blood-pressure was recorded after the intravenous injection of hypertonic sodium chloride solution, 39 were operated upon under

Table I.—NINETEEN CASES TREATED BY INTRAVENOUS INJECTION OF 30 PER CENT SODIUM CHLORIDE FOR SHOCK OCCURRING DURING OPERATION FOR INTESTINAL OBSTRUCTION PERFORMED UNDER SPINAL ANÆSTHESIA

Case No.	Sex and Age	Initial Blood-pressure	Systolic Blood-Pressure before NaCl	c.c. of 30 per cent NaCl	Systolic Pressure			Remarks
					5 min. after	10 min. after	30 min. after	
2	F. 60	140/100	70	40	120	130	140	Gum saline followed on
6*	M. 75	110/60	zero	50	100	—	140	Coramine and pituitrin: no action in 25 minutes
7*	M. 59	140/100	60	40	105	110	120	Blood-sodium during shock = 311 Blood-chlorine during shock = 347
9*	M. 52	170/100	85	40	160	180	140	Blood-sodium during shock = 302 Blood-chlorine during shock = 385
10	F. 46	130/80	80	40	100	100	80	Blood-sodium during shock = 344
12	F. 22	115/90	50	30	70	70	—	Evipan-sodium anæsthesia in addition
14*	M. 73	105/70	105	30	150	125	140	Injection made before spinal anæsthesia
15*	F. 54	160/100	50	80 15%	60	60	—	Responded to icoral rather than to NaCl
			60	40 30%	80	70	—	
21*	F. 30	115/80	60	30	100	100	—	Shock developed towards end of operation
22	F. 76	140/80	zero	40	80	50	—	Died 2 hours later
27	M. 73	145/80	60	50	80	80	—	Died 6 hours later
30*	M. 32	140/100	70	40	100	120	80	Followed by gum saline
32	F. 83	140/90	60	40	120	200	150	Combined action with icoral previously injected but which had shown no effect in 5 minutes
33	M. 53	120/65	50	40	—	100	120	
36	M. 21	120/70	80	40	110	115	100	
37*	M. 54	145/80	70	40	110	125	—	Previous injection of 40 c.c. 50 per cent glucose
38*	F. 52	160/100	zero	40	100	110	100	
39*	F. 49	120/70	95	40	150	175	—	Previous injection of 80 c.c. 50 per cent glucose
48	F. 69	130/70	60	40	100	110	100	

* Detailed chart of systolic and diastolic blood-pressure given. The pulse-rate record has been omitted for the sake of clarity.

spinal anæsthesia, and 19 of these were for intestinal obstruction. There were 6 operations for cerebral decompression under nitrous oxide and oxygen anæsthesia, 4 other cases under gas and oxygen, and 2 under local anæsthesia. There were 2 post-operative cases. The records will be considered in these groups for better comparison.

Among the group of operations for intestinal obstruction, in 3 cases (*Cases* 7, 9, and 10) blood was taken for the estimation of sodium and chlorine at the time when the blood-pressure was low. The figures obtained were 311, 302, 344 mgrm. sodium per 100 c.c. serum respectively. It was hoped to have continued these investigations with estimations of hæmoglobin, blood-urea, and blood-sugar, but practical difficulties make it impossible at present.

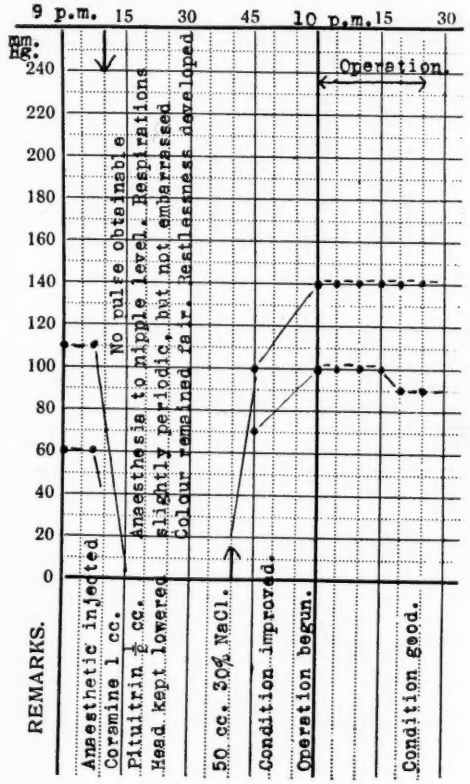


FIG. 81.—*Case* 6. Male, aged 75. Gall-stone ileus.

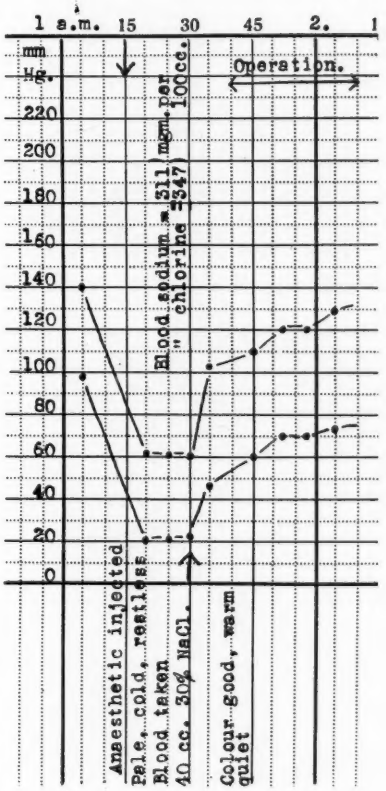


FIG. 82.—*Case* 7. Male, aged 59. Adhesions.

Case 6 is of interest in that 2 c.c. of racedrin given with the spinal anæsthetic and 1 c.c. of coramine and ½ c.c. of pituitrin had no effect even after twenty minutes, but 50 c.c. of 30 per cent sodium chloride raised the systolic pressure from zero to 100 mm. Hg in five minutes, with rapid recovery of the patient. The anæsthesia in this case was to a high level; presumably the hypertonic saline mobilized fluid from the paralysed muscles.

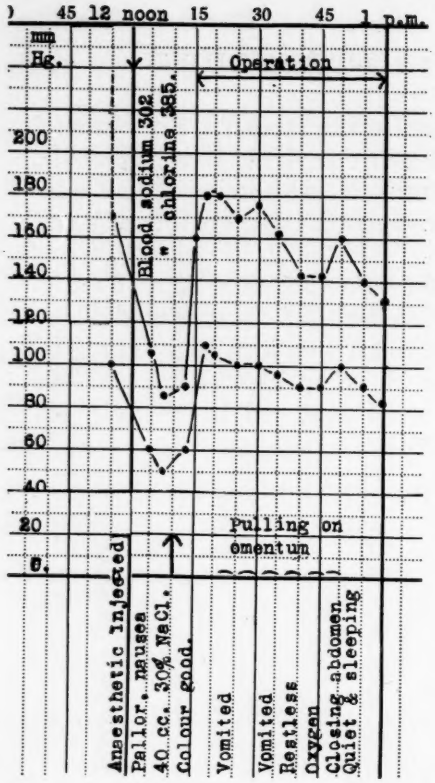


FIG. 83.—*Case* 9. Male, aged 52. Adhesions.

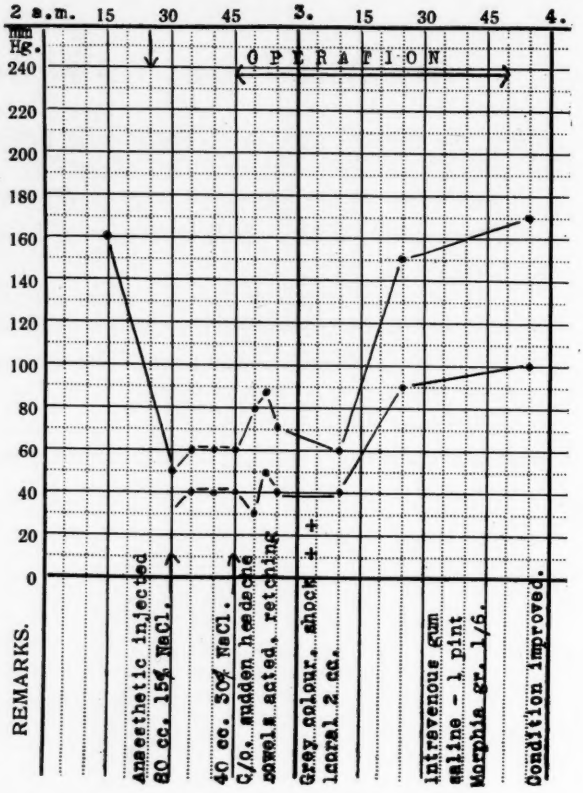


FIG. 85.—*Case* 15. Female, aged 54. Entero-anastomosis.

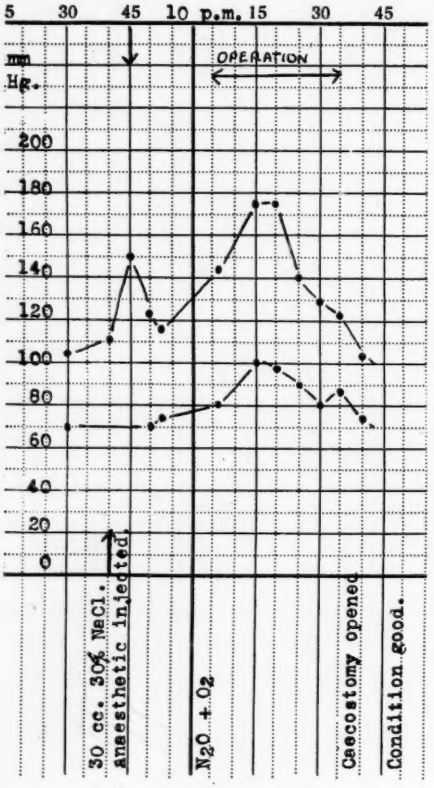


FIG. 84.—*Case* 14. Male, aged 73. Cæcostomy for carcinoma of colon.

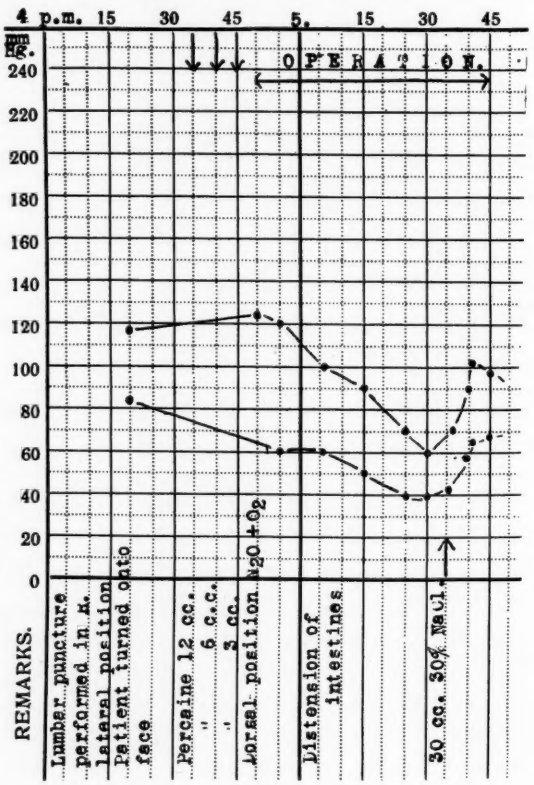


FIG. 86.—*Case* 21. Female, aged 30. Cæcostomy for carcinoma of colon.

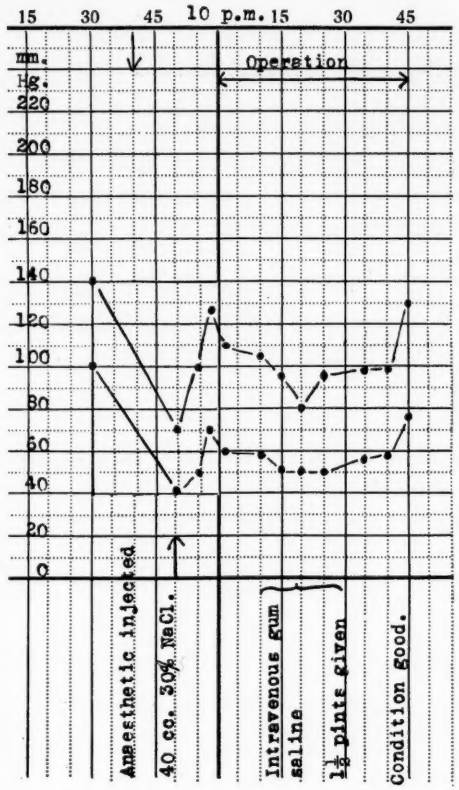


FIG. 87.—*Case* 30. Male, aged 32. Ileostomy for intussusception.

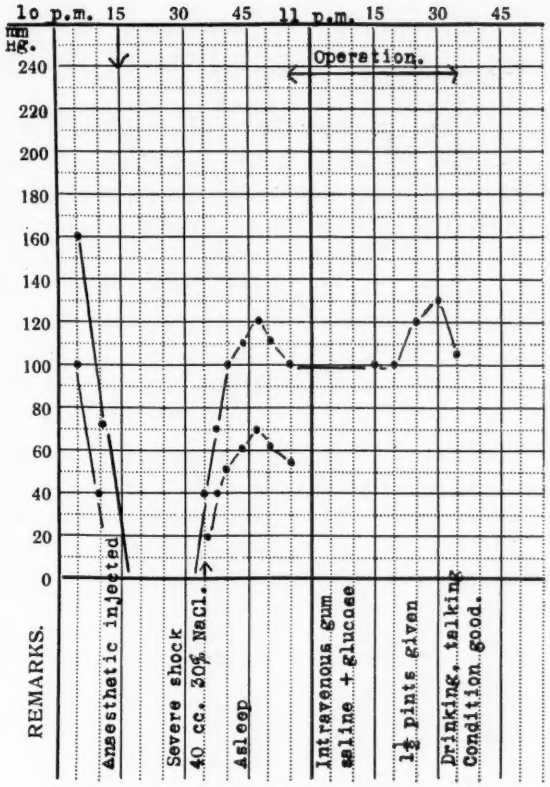


FIG. 89.—*Case* 38. Female, aged 52. Strangulated inguinal hernia.

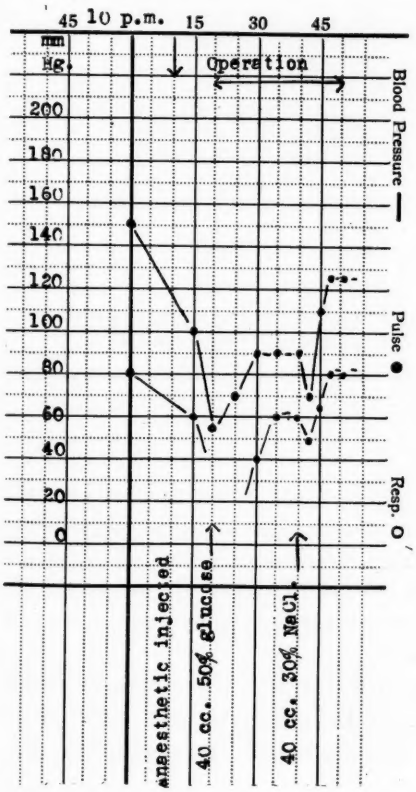


FIG. 88.—*Case* 37. Male, aged 54. Strangulated inguinal hernia.

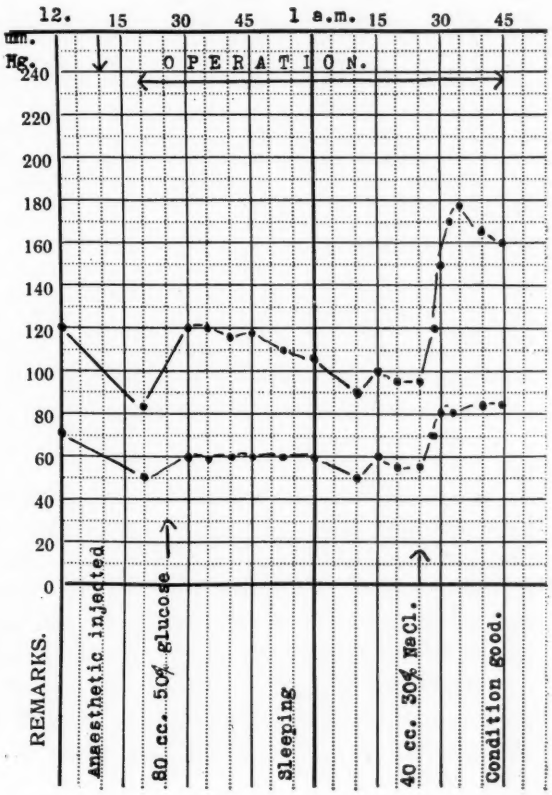


FIG. 90.—*Case* 39. Female, aged 49. 1st stage Mikulicz operation for carcinoma of colon.

Case 14 shows the effect of gas and oxygen in raising the blood-pressure.

Case 15 shows that 80 c.c. of 15 per cent sodium chloride had very little effect; 40 c.c. of 30 per cent raised the systolic pressure by 30 mm. Hg by its greater osmotic pressure, but in this case a cardiovascular stimulant was needed and 2 c.c. of icoral restored the blood-pressure to its normal level.

Cases 37 and 39 show the less powerful effect of a non-electrolytic solution even when hypertonic; 40 c.c. and 80 c.c. of 50 per cent glucose were injected intravenously, but neither raised the blood-pressure to the same level as 40 c.c. of 30 per cent sodium chloride which was given later.

Table II.—Twenty Cases of Shock occurring during Spinal Anæsthesia treated by Intravenous Injection of 30 per cent Sodium Chloride Solution

Case No.	Sex and Age	Initial Blood-pressure	Systolic Blood-pressure before NaCl	c.c. of 30 per cent NaCl	Systolic Pressure			Remarks
					5 min. after	10 min. after	30 min. after	
3	M. 66	120/60	80	20	105	105	—	Perforated gangrenous appendix
4	M. 41	140/80	50	20	85	85	—	Pyelolithotomy
5*	F. 54	140/80	100	30	180	175	140	Diverticulitis
11*	F. 5	130/80	50	15	95	120	95	Case of adrenal hyperplasia
			40	20	80	110	110	
16	F. 76	130/70	zero	50	80	120	—	Diathermy excision of vesical growth
18	F. 48	120/80	70	40	95	110	95	Large ovarian cyst
20	F. 59	100/80	45	20	90	85	70	Mickulicz operation, carcinoma colon
23	M. 35	130/80	85	40	100	130	—	Gangrenous appendix plus bronchitis
24	M. 24	150/90	90	40	110	130	145	Gangrenous appendix
25	M. 51	120/90	60	40	80	120	—	Appendicostomy for colitis
28	M. 53	120/70	70	40	110	150	140	Mikulicz operation, carcinoma colon
29	M. 71	100/60	90	40	140	125	—	Amputation leg—diabetic gangrene
42	F. 69	155/90	80	40	90	150	125	Colectomy for carcinoma
43	M. 34	120/80	95	40	135	145	130	Colectomy for carcinoma
44	M. 51	160/80	60	40	100	120	—	Perforated diverticulitis
45	F. 54	120/80	50	40	100	110	80	Gastro-enterostomy—carcinoma pancreas
46	M. 58	125/80	90	40	100	120	115	Excision of rectum
51*	M. 43	110/60	80	40	138	118	105	Partial gastrectomy
			80	40	120	120		
52	M. 39	160/110	75	40	125	125	130	Large adherent hydronephrosis
53*	M. 56	160/100	70	40	120	160	140	Cholecystectomy—jaundice

* Detailed chart of systolic and diastolic blood-pressure given. The pulse-rate record has been omitted for the sake of clarity.

Cases 11 and 51 both show that a second injection of hypertonic saline will have a similar effect on the blood-pressure. In each case after the first administration the blood-pressure remained steady until a further noxious stimulation caused further shock; by altering the osmotic pressure apparently fluid is restored from the tissue spaces into the circulation and after some sort of adjustment tends to return to normal and not to the shock condition.

The little girl aged 5 (*Case* 11) was of peculiar interest. She was explored for a possible adrenal tumour as she showed signs of virilism. Actually hyperplasia was present and one gland was removed. Each time the blood-pressure fell

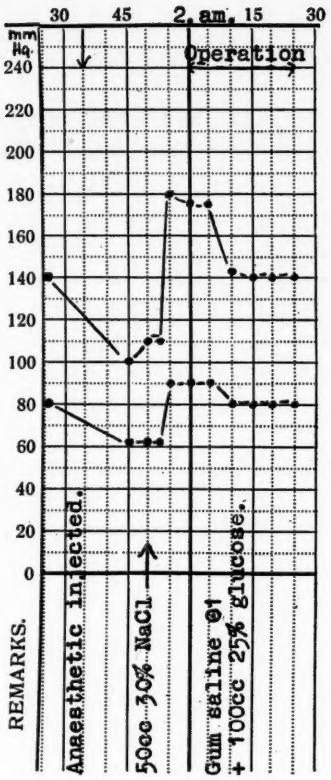


FIG. 91.—*Case* 5. Female, aged 54. Diverticulitis.

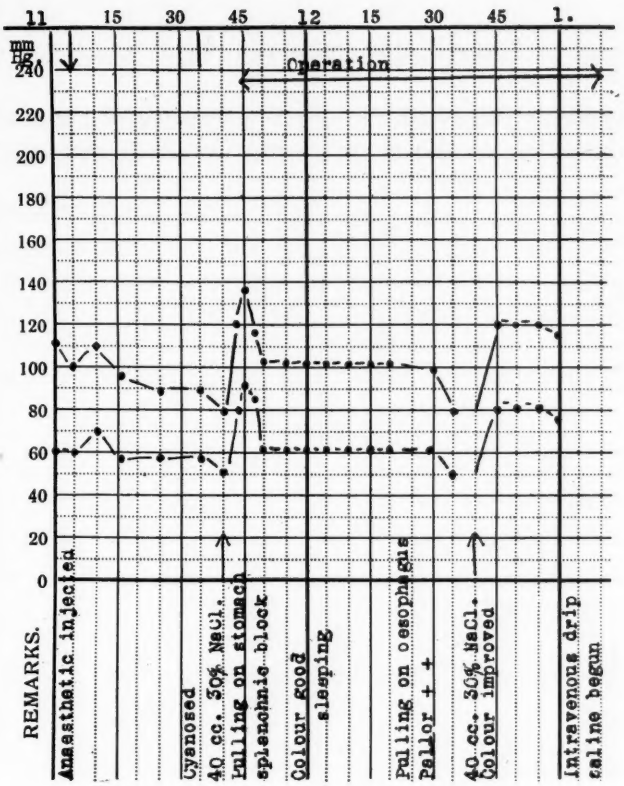


FIG. 93.—*Case* 51. Male, aged 43. Partial gastrectomy.

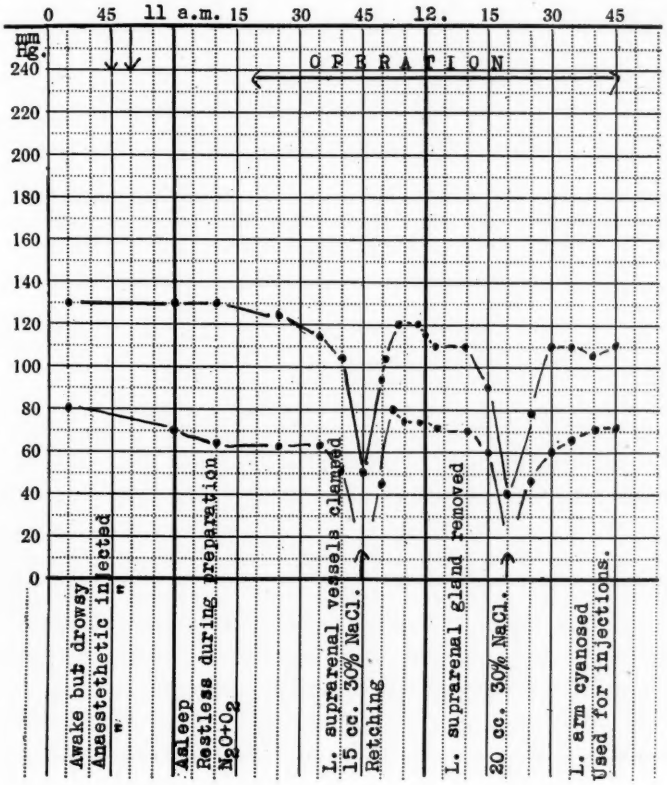


FIG. 92.—*Case* 11. Female, aged 5. Exploration—hyperplasia of adrenals.

suddenly there had been interference with the adrenals. There was gross paralytic distension of the stomach and intestines and neither the spinal anæsthetic nor

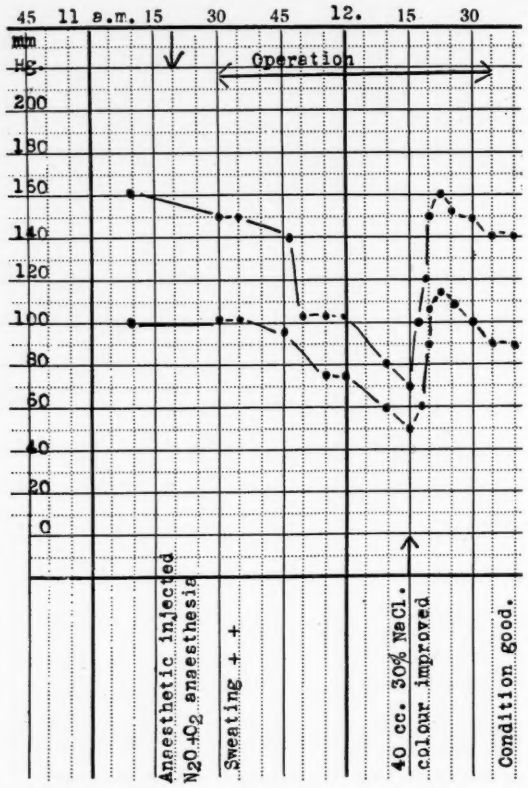


FIG. 94.—*Case* 53. Male, aged 56. Cholecystectomy. Stone in common duct. Jaundice.

Table III.—THE EFFECT OF 30 PER CENT SODIUM CHLORIDE INJECTED INTRAVENOUSLY TOWARDS THE END OF OPERATIONS FOR CEREBRAL DECOMPRESSION, WHERE THE INTRACRANIAL TENSION WAS RAISED

CASE NO.	SEX AND AGE	DURATION OF OPERATION BEFORE INJECTION	BLOOD-LOSS	BLOOD-PRESSURE BEFORE INJECTION	C.C. 30 PER CENT NACL	BLOOD-PRESSURE			REMARKS
						5 min. after	10 min. after	30 min. after	
		Hr. Min.							
1	M. 68	2 25	Slight	130/90	30	160/100	140/90	140/100	Left frontal tumour
34	F. 41	2 40	+ +	80/55	50	138/80	140/90	130/90	VIIIth nerve tumour
40	M. 29	3 10	+	85/40	50	140/70	140/70	100/60	Traumatic orbital meningocele
47	M. 52	1 35	—	80/55	40	118/80	118/80	—	Right frontal 2ndy. tumour with arterial hypertension
49	M. 52	1 10	—	85/?	40	105/80	125/85	140/95	Right frontal meningioma with arterial hypertension
50	F. 44	2 0	—	160/110	40	185/110	190/118	—	Right temporal lobe tumour

hypertonic saline had any effect in lessening it. Her pulse-rate was rapid, nearly 160, most of the time she appeared to be under predominant sympathetic stimulation.

Of interest in the group of cases in *Table III* is the fact that the same rise in blood-pressure occurs on injecting hypertonic saline even after a considerable loss of blood. In *Case* 34 a blood transfusion had just been begun when the injection was made, and in *Case* 40 gum saline was being given. In neither case had the infusions raised the low blood-pressure. In *Case* 50, where the blood-pressure was already high, a further increase occurred. There appeared to be no diminution in the intracranial tension.

Table IV.—HYPERTONIC SALINE GIVEN IN CASES OF SHOCK OCCURRING DURING OPERATIONS PERFORMED UNDER: (1) NITROUS OXIDE AND OXYGEN ANÆSTHESIA; (2) LOCAL ANÆSTHESIA

CASE NO.	SEX AND AGE	DURATION OF OPERATION BEFORE INJECTION	BLOOD-LOSS	BLOOD-PRESSURE BEFORE INJECTION	C.C., 30 PER CENT NaCl	BLOOD-PRESSURE			REMARKS
						5 min. after	10 min. after	30 min. after	
1. Nitrous Oxide and Oxygen Anæsthesia.—									
		Hr. Min.							
17	M. 31	1 50	+	90/50	50	100/50	118/60	100/50	Forequarter amputation for sarcoma left axilla
19	M. 61	1 0	—	60/45	40	100/60	130/80	—	Jejunal ulcer
26*	F. 48	—	—	40/?	40	140/100	180/120	140/90	Acute pancreatitis, shock++, pre-anæsthetic injection of NaCl
31	F. 62	1 0	—	80/50	40	120/60	140/90	110/70	Ileostomy
2. Local Anæsthesia.—									
8	M. 72	—	—	110/80	20	140/90	138/85	—	Cæcostomy, local anæsthesia, arteriosclerosis++
13	M. 75	—	—	80/70	40	120/70	130/70	50/?	Extreme collapse, strangulated umbilical hernia, drained

* Detailed chart of systolic and diastolic blood-pressure given. The pulse-rate record has been omitted for the sake of clarity.

Case 26, a woman of 46 suffering from acute pancreatitis, was severely shocked and restless. In spite of morphia and a pint of gum saline solution given intravenously before there was any interference, her systolic blood-pressure was only 40 mm. Hg. There was an immediate response to 40 c.c. of 30 per cent sodium chloride which enabled laparotomy to be performed.

Two post-operative cases were given hypertonic saline intravenously. *Case* 35, a woman of 40, was shocked following vaginal hysterectomy by a reactionary intraperitoneal hæmorrhage. Her pulse could not be felt and she responded to no treatment for three or four hours, when recovery gradually came spontaneously. The only time her pulse was felt during this period was immediately after injecting 40 c.c. of 30 per cent sodium chloride. *Case* 41 was a man of 37 with paralytic

ileus due to general peritonitis from appendicitis; 80 c.c. of 15 per cent sodium chloride were given intravenously to stimulate peristalsis. It failed to do this, but the blood-pressure rose from 130/80 to 150/90 in five minutes.

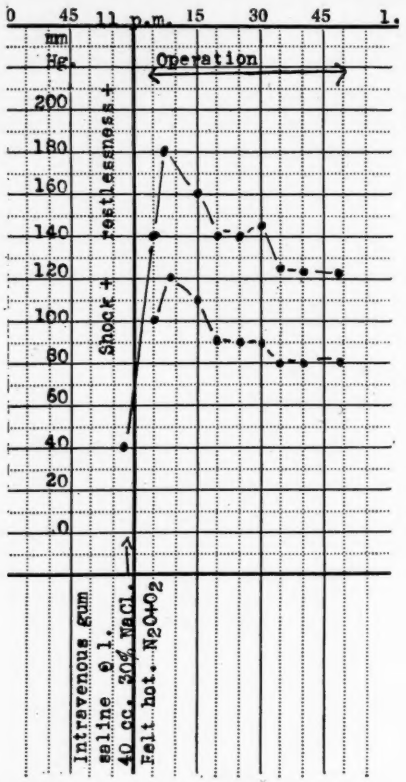


FIG. 95.—*Case* 26. Female, aged 48. Acute pancreatitis.

SUMMARY

1. The therapeutic uses of hypertonic sodium chloride solution are mentioned.

2. The advantages and dangers of spinal anæsthesia with special reference to its use in cases of intestinal obstruction are discussed.

3. The type of shock present in spinal anæsthesia is considered to be similar to the experimental traumatic shock studied in animals where the blood stagnates in the injured limbs, and in spinal anæsthesia in the anæsthetized and paralysed regions. Plasma escapes into the interstitial spaces, and the circulatory collapse may be comparable with that seen in Addison's disease.

4. The administration of vasoconstrictor drugs to raise the blood-pressure when shock has developed may be ineffectual and undesirable on account of the further reduction of the oxygen supply to the tissues and vital organs.

5. Extra fluid introduced into the circulation is good pre-operative treatment in cases of intestinal obstruction where there is dehydration, but may not prevent the fall in blood-pressure caused by the onset of spinal anæsthesia.

6. In order to mobilize fluid from the tissue spaces, hypertonic saline solution (usually 40 c.c. of 30 per cent sodium chloride) has been injected intravenously.

The circulation was stimulated immediately, with improvement in the patient's condition. In five minutes a considerable increase in blood-pressure could be recorded. The rise was towards the level normal for the individual. Hypertension was not found, as may occur when using the more potent vasoconstrictor drugs. After twenty to thirty minutes when the body fluids had become re-adjusted the blood-pressure readings were on a lower level, but no longer at a shock level unless this was freshly induced by the operative procedure.

The intravenous injection of a small quantity of strongly hypertonic sodium chloride solution appears to be a reasonable method of treating the low blood-pressure occurring at the onset of spinal anæsthesia, and has been found of practical benefit to the patient in tiding over a period of crisis.

My thanks are due to the surgeons of the Leicester Royal Infirmary for their permission to inject hypertonic saline in these cases.

REFERENCES

[1] WEED, " Experimental Alterations in Brain Bulk ", *Amer. Jour. Physiol.*, 1919, xlviii, 512, 531.
[2] OCHSNER, " Motility of the Intestine ", *Arch. of Surg.*, 1933, xxvii, 742.
[3] ROGERS, *Tropical Medicine*, 1935, 290. London : Churchill.
[4] GRAHAM, *St. Bart.'s Hosp. Rep.*, 1929, lxii, 55.
[5] BAYLISS, *Intravenous Injection of Wound Shock*, 1918. London : Longmans, Green & Co.
[6] SWINGLE and PFIFFNER, *Amer. Jour. Physiol.*, 1933, 108–144.
[7] LOEB, ATCHLEY and STAHL, " Role of Sodium in Adrenal Insufficiency ", *Jour. Amer. Med. Assoc.*, 1935, civ, 2149, 2177.
[8] MACFEE and BALDRIDGE, " Infusion Treatment of Shock ", *Ann. of Surg.*, 1934, c, Aug., 266.
[9] CUSHING and FOLEY, *Proc. Soc. Biol. and Med.*, 1920, xvii, 217.
[10] QUARELLA, " Spinal Anæsthesia with Percaine ", *Minerva Med.*, 1930, Sept.
[11] SEBRECHTS, Technique described by J. W. West, *Jour. R.A.M.C.*, 1933, July.
[12] JONES, HOWARD, *Brit. Jour. Anæsth.*, 1930, vii, April and July.
[13] BLALOCK, " Cause of Low Blood-Pressure Produced by Muscle Injury ", *Arch. of Surg.*, 1930, xx, 959.
[14] HOLT and MACDONALD, " Observations on Experimental Shock ", *Brit. Med. Jour.*, 1934, i, 1069.
[15] HUGHES, *Ibid.*, 1935, 2, 3.
[16] MONCRIEFF, " Respiratory Failure ", *Lancet*, 1935, ccxxviii, 664.
[17] FLINT, " Experiences with Spinal Anæsthesia ", *Brit. Med. Jour.*, 1935, i, 197.
[18] BOYCOTT, " Regulation of Blood Volume in Normal and Nephritic Animals ", *Jour. Pathol. and Bacteriol.*, 1913, xviii, 11.
[19] SMITH and MENDEL, " Adjustment of Blood Volume after Injection of Isotonic Solutions ", *Amer. Jour. Pathol.*, 1920, liii.
[20] ENGELS, *Arch. f. exper. Pathol. u. Pharmakol.*, 1904, li, 346.
[21] PETERS and VAN SLYKE, *Quantitative Clinical Chemistry Interpretations*, 1931, i, 665, 1034, 1021. Baltimore : Williams and Wilkins.
[22] BARBER and ORIEL, " Allergy ", *Lancet*, 1928, ccxv, 1009.
[23] ANDREWS, " Experimental Uræmia ", *Arch. of Internal Med.*, 1927, 548.
[24] WEED, *Physiol. Rev.*, 1922, ii, 171.

SOME OBSERVATIONS ON THE SURGERY OF TRIGEMINAL NEURALGIA

By E. P. STIBBE

READER IN ANATOMY, KING'S COLLEGE, LONDON

THE first intracranial operation for the relief of idiopathic trigeminal neuralgia was performed in this country by Victor Horsley[1] in 1891. Horsley approached the nerve by the intradural parietal route and resected a portion of the Gasserian ganglion. The patient died, though post-mortem examination showed that there was no gross brain-lesion.

Between 1891 and 1901 there were sporadic reports of cases operated on by this method; Tiffany,[2] for example, fully reports a successful case.

In 1901 Frazier[3] introduced the extradural method of approach, and he replaced gasserianectomy by partial resection of the sensory root of the nerve. This gradually became the standard method and is the basis of the practice of most neurological

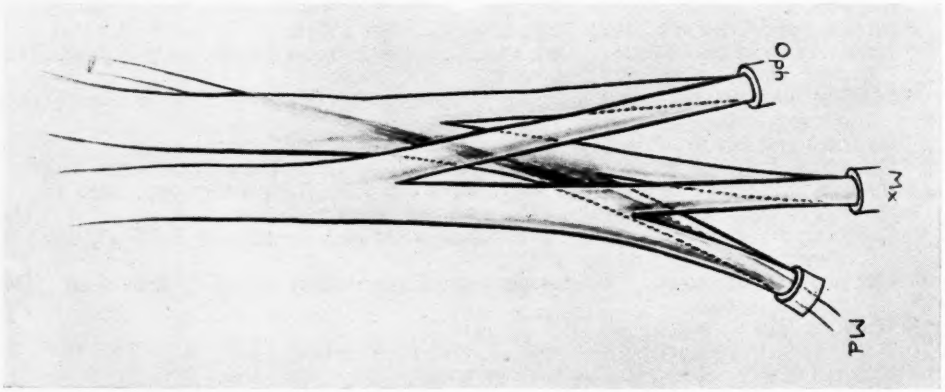


FIG. 96.—To illustrate Dandy's postulate of the disposition of the fibres of the sensory root of the trigeminal nerve. Oph., Ophthalmic division; Mx., Maxillary division; Md., Mandibular division.

surgeons in this country. In 1925, however, Dandy[4] introduced the method of intradural approach to the nerve by the posterior fossa and, with the new method, he offered several criticisms of the old. He held that the Frazier method must cause anæsthesia as well as analgesia, and claimed that by section of the sensory root close to the pons instead of (as previously) close to the ganglion, he produced analgesia without anæsthesia. In explanation of this result Dandy postulated (though he did not pretend to have proved) a possible re-arrangement and segregation of the fibres in their passage from the ganglion to the pons (*Fig.* 96). Thus the pain-fibres of each division of the nerve separate off from the touch-fibres soon after leaving the ganglion, and come to lie in the posterior (or lateral) half of the nerve; while the touch-fibres similarly collected together occupy the anterior (or

medial) half of the nerve as they approach the pons. This hypothesis would readily explain the difference between the results of the two methods. By dividing about the outer one-third of the nerve close to the ganglion, one would produce analgesia and anæsthesia in the mandibular division. By dividing about the outer one-half of the nerve close to the pons, one would produce general analgesia of all three divisions, but no anæsthesia.

Before reporting the investigations which form the subject of this paper, reference must be made to our previous knowledge of the anatomy of the Vth nerve and its nuclei. With regard to the nuclei it is certain that the principal nucleus is a relay station on the path of epicritic or discriminative fibres, and that the descending or spinal root, relayed in the spinal nucleus, carries pain and thermal impulses. The best evidence for this is the dissociation of symptoms into loss of touch-sensation and loss of pain-sensation seen as the result of lesions involving only one of these two nuclei. Stopford[5] summarizes the evidence and illustrates the paths of the fibres in his book, and in papers on the function of the spinal nucleus and on the posterior inferior cerebellar arteries. Many other writers—for example, Gillis,[6] W. Harris,[7] and M. Gerard,[8] report individual cases of lesions of the spinal nucleus producing dissociation of sensation (the usual lesion is thrombosis of the posterior inferior cerebellar artery); Stopford[9] has collected these and added them to his own; and he concludes that the evidence is ample to prove the protopathic function of the spinal fibres and nucleus.

There is no uniformity of opinion on just what the fibres of the sensory root of the Vth nerve are like, or on the details of their fate before or after their entry into the pons. In text-books[10] a general description is given of the fibres of the sensory root as ' dividing into two branches, of which the upper go to the principal nucleus while the lower form the descending or spinal root.' This would imply that protopathic and discriminative impulses are carried by one fibre, and that the sensations only take separate paths when they reach collaterals of the fibre; this conception presents great difficulties. Fortunately Gudden[11] describes undivided fibres to a considerable number amongst the divided fibres (about the histological existence of which there is no doubt). So we may suppose on the one hand that some fibres do actually go to both nuclei, and that the difference lies perhaps in threshold of response to stimulation; but that others go only to one nucleus or the other.

Mention should be made of Ranson's argument[12] on physiological grounds, that pain-fibres should be unmyelinated. This may be dismissed for our purposes, since (as shown, for example, by S. L. Koch[13]) the number of non-medullated fibres in the human Vth nerve is very small.

We may take it, then, that while the anatomical observations are not very precise or convincing, the fibres within the pons go to one or other of the two nuclei under discussion.

Kappers,[14] in the course of his most illuminating work on the comparative anatomy of the nervous system of Vertebrata, works out the functions of the different central parts of the Vth nerve. Thus the spinal root and nucleus are the oldest part of the nerve phylogenetically, and in animals below the Reptilia subserve the reflexes of the skin of the head. Only in certain reptiles does the anterior end of the spinal nucleus enlarge and begin to become an entity (principal nucleus), and thus the newer epicritic or discriminatory paths begin. So we may picture the principal nucleus and trigeminal fillet as a contribution to the development of the

neopallial system and the spinal nucleus as the original archepallial non-discriminative system.

For these reasons we may take as a working basis the conception that *somewhere between the Gasserian ganglion and the actual nuclei* the sensory fibres of the Vth nerve undergo a rearrangement and a segregation into discriminatory touch-fibres (principal nucleus) and protopathic fibres (spinal nucleus).

Dandy's hypothesis, then, is only a slight addition to the known facts; it is certain that functional segregation takes place, and Dandy suggests that it takes place in the nerve outside the pons.

There is some clinical evidence in support of this view. Careful examination of a large number of cases of sensory disturbances due to mechanical involvement of the Vth nerve by tumours of the posterior fossa shows that pain sense is lost and tactile sense unimpaired, or relatively unimpaired. A typical case of involvement of the Vth nerve by an acoustic tumour shows a sense of numbness, and definite loss of sensibility to pain; but a pinprick is quite well orientated, but with sensation of touch only. Moderate degrees of heat and cold give variable results, but touch to cotton-wool is definitely appreciated and orientated. Further, the loss of pain sense follows the order one would expect according to Stopford's investigations[15] —namely, that the supra-orbital region is first affected, then the cheek, and lastly the mandibular region—corresponding to the known order of fibres from behind forwards. In one case actually a decompression operation (the tumour being deemed irremovable) led to the disappearance of the symptoms in the reverse order —namely, mandibular, buccal, and, lastly, forehead.

These considerations justify the expectation that a section of the outer part of the nerve close to the pons would destroy protopathic fibres, but a similar section close to the ganglion would destroy a topographic portion of the nerve—i.e., some protopathic and some discriminatory fibres arising in a particular region of the face. If these two operations are done experimentally, the first (near the pons) should result in complete degeneration of the spinal root of the nerve; the second (near the ganglion) should result in partial degeneration of the spinal root and partial degeneration of the fibres around the principal nucleus.

Accordingly two series of experiments have been carried out—partial section of the sensory root of the trigeminal nerve by the parietal route, the nerve being cut close to the Gasserian ganglion; and partial section by the posterior-fossa route, the nerve being cut close to the pons.

TECHNIQUE AND RESULTS

Well-grown Macacus monkeys were anæsthetized by induction with C.E. mixture, followed by introperitoneal dial (1½ c.c.) or nembutal (4 gr.). (Nembutal acts so quickly that the general anæsthetic may be stopped immediately after the injection.)

Method 1.—Frazier's transparietal extradural, with distal section of the nerve (outer one-third of fibres). Four successful operations. Two killed on eleventh and thirteenth day respectively; medulla, pons, and lower midbrain prepared by Marchi's method. *In one case no degeneration at all, in the other so few and scattered fibres as to be of doubtful significance.* The other two were killed on the twentieth and twenty-eighth day respectively and the brain-stem prepared by Weigert Pal. Again *no degeneration found sufficient to be assessed.*

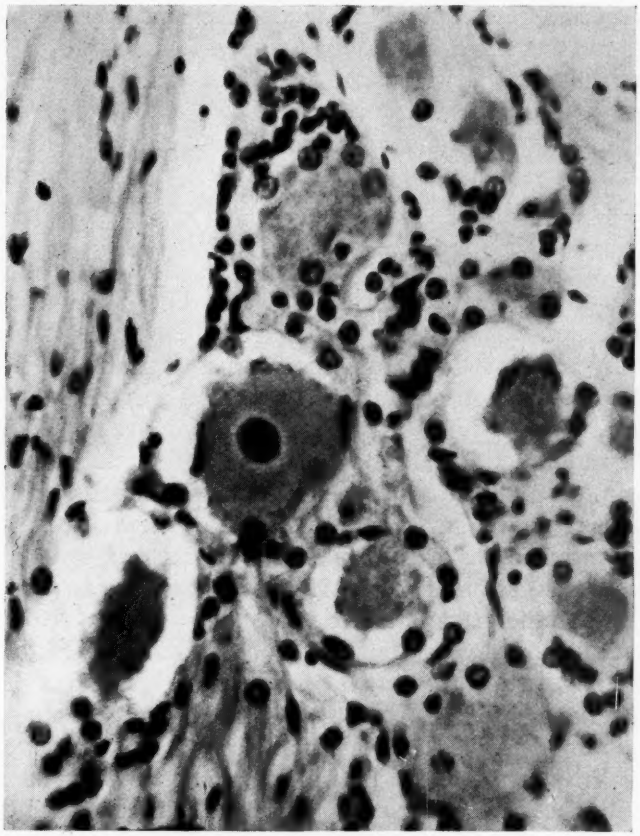


FIG. 97.—Sensory cells in sensory root of trigeminal nerve near pons. Macacus. H.E. (× 720.)

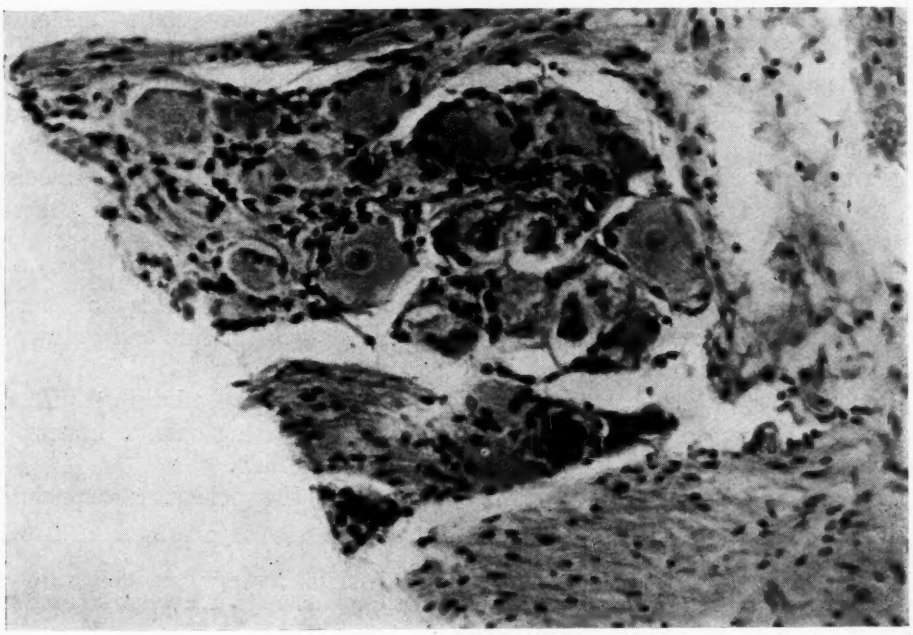


FIG. 98.—Sensory cells in sensory root of trigeminal nerve near pons. Human. H.E. (× 360.)

These unexpected results were so disconcerting as to make one feel it was necessary to revise one's technique. The explanation, however, came to light during the examination of the stumps of the nerve, which had been preserved. It was found (*Fig.* 97) that the sensory root contained considerable numbers of large sensory nerve-cells at various points of its course, quite separate from the Gasserian ganglion; generally these were in the central half of the root, often quite close to the pons. In the distal operation, therefore, the reason why little or no central degeneration was found, was that the fibres were cut peripherally to their cells of origin. Examination of the Vth nerve in man showed similar groups of centrally-placed cells* (*Fig.* 98). These cells must be regarded as of great significance. The conception that section of the sensory root of the nerve proximal to the ganglion ought permanently to throw it out of action is based on the view that the nerve fibres proximal to the ganglion are a part of the central nervous system and therefore do not regenerate. Actually a proportion of the fibres are cut distal to their cells and might regenerate peripherally. Such regeneration might be the cause of recurrence of pain. It must be added that recurrences nowadays are few, and only after long intervals; but we have, in the presence of these cells, an indication that recurrences are possible when the peripheral operation has been performed.

Method 2.—Dandy's intradural operation via the posterior fossa, with section of the outer half of the nerve close to the pons.

Three successful operations, but only one satisfactory histological result—namely, degeneration of approximately the whole of the spinal root of the nerve with no other degenerations (*Figs.* 99, 100). The reason why the other cases did not succeed histologically is that various other patches of degeneration were found, quite remote from the Vth-nerve tracts. This is due to damage by torsion of the brain-stem during the displacement of the cerebellum; the one successful case was a lucky one in which this had not occurred, but the result is so clear-cut that the writer feels that even one such case is convincing.

Anatomical and histological evidence has been sought for—that is, demonstrations that the fibres do cross as the experimental work indicates. In this connection Frazier states definitely that the division of the nerve into three parts—ophthalmic, maxillary, and mandibular—is topographically represented in the ganglion and at least in the distal part of the sensory root. Whitehead[17] follows this up by demonstrating that in embryos the ganglion consists of three distinct parts each associated with its own topographic division of the nerve, and goes on to show that there is 'no tendency for the fibres of the sensory root to intermingle.' Woollard and Carmichael[18] state more guardedly that 'a study of the ganglion and the central root by anatomical methods has led to the conclusion that there is no complete segregation of fibres in the central root corresponding to epicritic and protopathic functions.'

Reports have also been made on the Vth nerve of animals other than primates. The present writer is not prepared to accept these as having definite bearings on the practical problem of the disposition of the fibres in man.

Dissections of the Vth nerve in man have proved disappointingly inconclusive,

---

* Since this work was done the writer has seen a paper by Peters[16] describing these same cells in various animals. Peters's publication was in October, 1935, the writer's observation approximately at the time of Peters's publication.

FIG. 99.—Right half of medulla of Macacus eleven days after resection of posterior third of sensory root of trigeminal nerve close to pons. Massive degeneration of spinal root of Vth. (Marchi.) (× 90.)

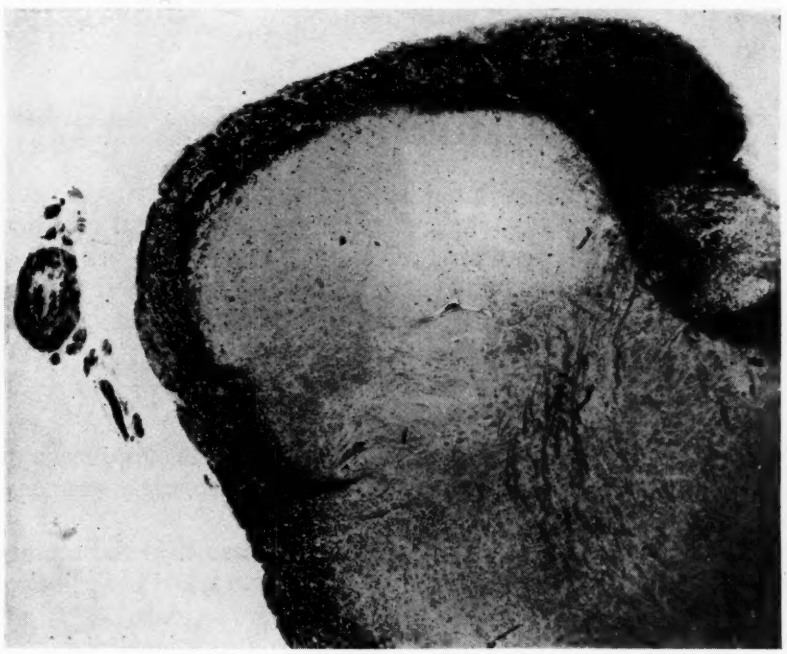


FIG. 100.—Control left side of medulla of *Fig.* 99. (Marchi.) (× 90.)

possibly owing to the failure so far to devise a technique. It is certain that the fibres of the sensory root do intermingle, crossing and recrossing; that fasciculi, jutting off, branch here and there, joining up with one another again, and splitting. The superficial attachment of the nerve to the pons is exceedingly fragile in virtue of the fact that immediately upon entering the pons the fibres break away to such an extent that so far no single convincing and complete dissection has been produced. On the other hand, these difficulties should not be insurmountable, and the imperfect dissections (*Fig.* 101) strongly suggest that the nerve close to the pons is segregated

FIG. 101.—Dissection of sensory root of trigeminal nerve. A, Protopathic fibres to spinal root; b, Fibres going mostly to principal nucleus; b', A few antero-medial fibres to spinal root.

into two parts: (1) Outer or posterior fibres which go entirely to the spinal root; and (2) Inner or anterior fibres, some of which go to the spinal root but most to the principal nucleus. It is proposed to follow this side of the work up by devising some method for fixing in situ before dissection. The histological structure of the nerve is also being studied and the results are being reported elsewhere; they should, when complete, offer useful collateral evidence.

SUMMARY

1. A brief history is given of the origin of two methods of approach to the Vth nerve for surgical resection in the treatment of trigeminal neuralgia—namely, Dandy's and Frazier's.

2. Experimental work on monkeys shows that there is in the sensory root of the Vth nerve a crossing and segregation of the fibres into spinal and principal nucleus fibres.

3. This division can properly be interpreted as a functional division into pain and touch fibres.

4. The sensory root of the Vth nerve contains numbers of large sensory nerve-cells, in scattered groups often quite close to the pons. Section of the nerve distal to these as practised by Frazier does not rule out the possibility of regeneration of the fibres.

REFERENCES

[1] HORSLEY, *Brit. Med. Jour.*, 1891, pt. 2, 1139.
[2] TIFFANY, *Ann. of Surg.*, 1894, xix, 47.
[3] FRAZIER, *Philadelphia Med. Jour.*, 1902, x, 594.
[4] DANDY, *Bull. Johns Hopkins Hosp.*, 1925, xxxvi, pt. 2, 105.
[5] STOPFORD, *Sensation and the Sensory Pathway*, 1930. London.
[6] GILLIS, *Jour. Amer. Med. Assoc.*, 1914, lxiii, 1550.
[7] HARRIS, W., *Proc. Roy. Soc. Med.* (Neurol. Sect.), 1909, ii, 77.
[8] GERARD, M., *Arch. Neurol. and Psychiat.*, 1923, ix, 306.
[9] STOPFORD, *Jour. of Anat.*, 1924–5, lix, pt. 2, 120.
[10] GRAY, *Anatomy*, 20th ed., 984.
[11] GUDDEN, *Allg. Zeits. f. Psychiat.*, 1891, xlviii, 16.
[12] RANSON, *Brain*, 1915, xxxviii, 487.
[13] KOCH, *Jour. Comp. Neur. and Psych.*, 1916, xxvi, 541.
[14] KAPPERS, *Die vergleichende Anatomie der Nervensystem der Wirbelthiere und des Menschen*, 1920. Haarlem.
[15] STOPFORD, Loc. cit.
[16] PETERS, *Jour. Comp. Neurol.*, 1935, lxii, 349
[17] WHITEHEAD, *Brain*, 1925, xlviii, pt. 1, 458.
[18] WOOLLARD AND CARMICHAEL, *Proc. Anat. Soc.*, 1932, lxvii, 204.

RECONSTRUCTION OF THE COMMON BILE-DUCT

By J. HAROLD COUCH
DEPARTMENT OF SURGERY, UNIVERSITY OF TORONTO

THE children of Israel, when commanded by the Egyptians to make bricks without straw,[1] were in no more difficult position than the modern surgeon who is faced with the task of reconstructing the common bile-duct, particularly when the damage has been so extensive that the right and left hepatic ducts are represented only by two yawning black holes on the under surface of the liver. One of the most difficult technical procedures in the upper abdomen is the reconstruction of a damaged common bile-duct. The purpose of this paper is to describe a method of reconstruction in a specific case, and to discuss the principles which have contributed to a successful issue. So far as can be determined, some of the details herein described are unique.

Edmund Horgan,[2] in a very complete monograph, *The Reconstruction of the Biliary Tract*, provides 'a review of all the methods that have been employed', and lists, in this work, nearly every conceivable anastomosis between liver, hepatic ducts, cystic duct, gall-bladder, common duct, duodenum, stomach, jejunum, colon, and external fistula.

As Coors[3] observes, almost every type of short-circuit has been carried out. The gall-bladder has been anastomosed successfully to stomach, duodenum, jejunum, common duct, and colon. The cystic duct has been anastomosed to stomach, duodenum, jejunum, and colon. The common duct has been anastomosed to stomach, duodenum, and jejunum. The hepatic duct has been anastomosed to stomach, duodenum, and jejunum. Even denuded liver substance has been anastomosed to duodenum and jejunum. End-to-end anastomosis of damaged ducts has been done either with or without the use of the splinting aid of rubber tubing, T tubes, and L tubes. Implantation of an external biliary fistula has been successfully made into the stomach, duodenum, bile-duct, and jejunum.

Plastic reconstruction of portions of the bile-duct has been accomplished both by pedicle flaps from the stomach and longitudinal incisions sutured transversely. Strictures of the ducts have been successfully dilated by means of forceps and bougies. The above-mentioned anastomoses have been accomplished both by direct coaptation and also by the utilization of many ingenious intervening foreign materials, such as Murphy buttons, decalcified bone bobbins, and rubber tubing with clever cuffs upon which are superimposed rubber rings to aid their retention in the ducts for sufficient time to permit complete epithelialization of the newly formed canal.

Nowhere, however, does Horgan describe a procedure which corresponds exactly to that which we were compelled to carry out in this particular case. In the first place, all his operations on hepatic ducts are sufficiently low for the right and left hepatic ducts to have already united to form the common hepatic duct. He does not mention any operation on hepatic ducts which are so short that the right

and left sides are without any extra-hepatic tissue whatever. In the second place, he does not describe a four-way tube such as we devised, to effect the repair of this type of injury.

Since the gradual trend in favour of cholecystectomy as opposed to cholecystostomy, injury to the common duct is no longer rare, in spite of all the efforts to avoid it. There is no arbitrary best method of removing the gall-bladder. Perhaps in most cases where the removal is simple, it is best accomplished from below upward after dividing the cystic duct. In the very obese patient with a deep-seated gall-bladder and liver well up under the costal margin, removal from above downward may favour visualization and so be safer.

In any case, the one underlying principle most likely to safeguard the common duct from mechanical injury, though perhaps not from later fibrosis, is the unyielding insistence on adequate exposure and direct visualization of cystic, common, and hepatic ducts before dividing any duct structure at all.

The history of our patient may be briefly cited. Mrs. E. H., aged 40, gave a history of cholecystitis with biliary colic in 1910. She suffered from typical gall-bladder indigestion with occasional attacks of colic at widely spread intervals from 1910 to 1932. At this time her gall-bladder was removed with considerable difficulty owing to dense adhesions. The cystic duct was dilated to the size of the gall-bladder, and, being filled with large, neatly packed, faceted stones, so resembled the gall-bladder that it was removed completely. A portion of the common duct was either removed or so damaged as to be useless.

For six months following operation the patient was relatively comfortable and showed no external fistula. At the end of that time she suffered a recurrence of pain associated with fever, chills, and jaundice.

After routine pre-operative preparation by transfusion and intravenous calcium chloride, a further operation was carried out for the purpose of re-establishing the continuity of her common bile-duct. At the time of operation she was in only fair general condition. She had lost weight and was deeply jaundiced, with itching.

The operation was technically difficult owing to the dense adhesions present. With patience, however, a clear and adequate exposure was obtained, revealing, to our dismay, two large black holes on the under surface of the liver. Both the right and left hepatic ducts were dilated to about 1 cm. in diameter and there was no extra-hepatic duct tissue. The remnant of the common bile-duct was represented at the other end by a small channel 1 in. long which extended upward from the duodenum in the direction of the under side of the liver.

A catheter was introduced into both right and left lobes of the liver for a distance of about 6 in., and after the intrahepatic duct system was washed thoroughly with saline, a small amount of muddy bile was obtained. Again necessity became the mother of invention, and a large T tube, converted into a four-way tube, was placed in position. The method of modifying the T tube is best shown in *Figs.* 102, 103, and needs no further explanation.

The duodenum, together with the attached stump of common bile-duct, was drawn up as close as possible to the under side of the liver, and in this position was securely fastened around the neck of the four-way tube by means of interrupted sutures, all of which were placed in position before being tightened. The long end of the tube was brought to the surface through a separate stab-wound.

The abdominal wall was closed in layers, and the patient was returned to bed in only fair condition.

We may pause at this point to emphasize the value of a clean exposure of the whole field of operation before proceeding to any active repair operation. The time consumed in clearing the field gently and safely, yet thoroughly, may be much greater than that consumed in the actual repair, but it is time well spent, for by this means only is it possible for an accurate and gentle re-adjustment to be made. Furthermore, the situation is well in hand at all times, and the dangers of bleeding or damage to bowel or other tissue are thus minimized, a consideration of value in a jaundiced patient who bleeds easily.

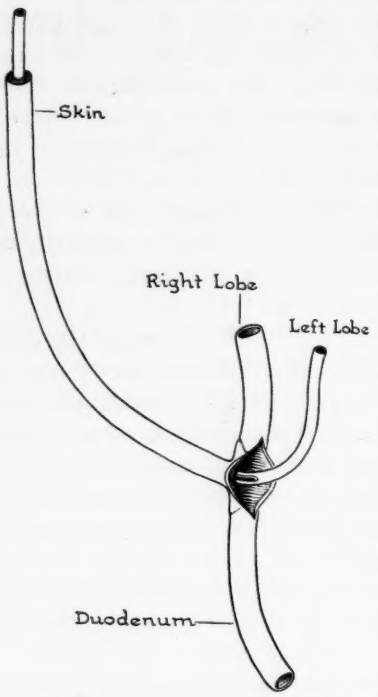


FIG. 102.—T tube converted into a four-way tube, showing the destination of its extremities.

The post-operative convalescence was decidedly stormy owing to the complication of continuous oozing of blood into the abdominal wall —a result, no doubt, of the jaundiced condition. Repeated transfusions were necessary in order to save the patient's life. Later, when the danger of death from hæmorrhage had been overcome, the convalescence was further complicated by loss of appetite consequent upon loss of bile to the surface.

Loss of appetite and loss of spirit are definite and well-recognized accompaniments to loss of bile, and can at times be cause for real concern. The patient may become depressed and melancholic. This fact again confirms ancient observations that the biliary flow has an influence upon moods. The evil effect of loss

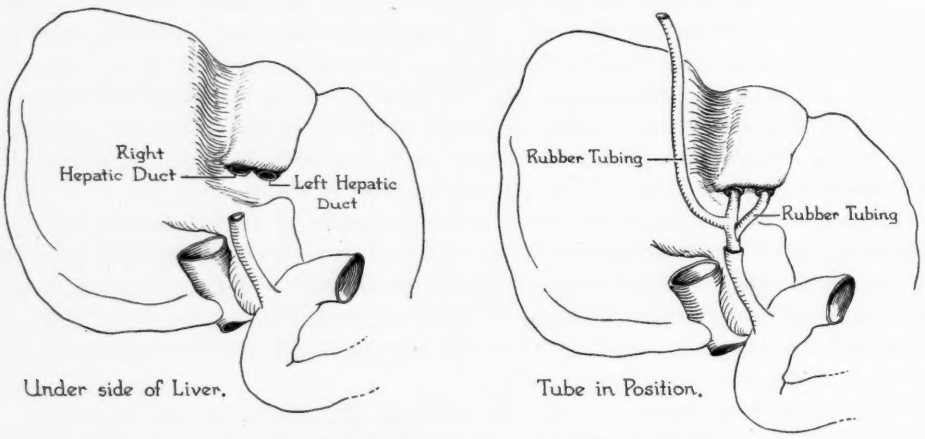


FIG. 103.—Showing the four-way tube fitted into position.

of spirit on both patient and surgeon is neatly circumvented to-day by the simple expedient of catching the patient's own bile in a bottle hung under the bed, and

feeding it back to him daily via the duodenal tube. The new lease of life resulting from the return of bile to the digestive tract has been the turning-point in more than one convalescence.

The four-way tube in this case was, however, kept clear by irrigation, and gradually the discharge to the surface diminished. The patient was then allowed to go home wearing a dressing pad over the end of the tube, which, two hours prior to each meal, was shut off, thereby forcing the bile into the duodenum.

The patient continued at home to gain in health and spirit. Her appetite improved, her urine and bowel movements were normal in colour, and the jaundice disappeared. Accordingly, she was allowed to resume her household duties for a period of eight months, at the end of which time she was re-admitted.

After the injection of thorotrast through the tube, the condition of her biliary system was carefully investigated by means of X rays.

Thorlakson and MacMillan,[4] by demonstrating the changes which occur, have confirmed the surgical observation that these tubes should remain in position for a very long time. They point out that the dilatation of the intrahepatic biliary tract, as a result of calculus, cicatrical, or neoplastic obstruction, produces a state of affairs within the liver quite comparable to bronchiectasis within the lung, and call this dilatation ' cholangiectasis '. The damming back of bile produces : cylindrical dilatation of the common duct and hepatic ducts ; sacculation of the intrahepatic biliary system ; pressure atrophy of the parenchymatous liver cells ; interference with the portal circulation ; cholangitis ; hepatitis ; and, finally, fibrosis of the whole liver with impairment of its function.

They show these structural changes radiographically, and demonstrate the improvement which follows prolonged drainage.

The degree of dilatation and damage is said to correspond with the severity of infection and duration of the obstruction. The ratio influences, therefore, the length of time necessary for drainage. If lipiodol or thorotrast is injected through the T tube, some will find its way into the duodenum. Most, however, will pass into the intrahepatic duct system to its finest radicles although there is no obstruction at the duodenum. Failure to visualize the intrahepatic system indicates, therefore, a widespread cholangitis, which will subside following prolonged drainage.

The technique of this investigation is simple : 20 c.c. of thorotrast are injected, plates are made immediately, and again at the end of twenty minutes. Dilatation of the main duct, sacculation of the lesser ducts, or delay in emptying are indications that the liver and duct system have not recovered. Drainage, then, must be continued. When the liver tree is normal and drainage prompt, the tube may be removed. By this method, it is possible also to prove free flow of bile through the ampulla of Vater. While it is true that others[5-8] had previously injected opaque materials into the biliary system, Thorlakson and McMillan were the first to pursue this study in a consistent manner, and to correlate X-ray findings within the tree with the physical condition of the patient. It is a well-known clinical observation that the T tube must be left in for a long time, but we have, in this method of investigation, a means of indicating when the tube may be removed, by the careful measurement of the improvement taking place in the liver.

When it was demonstrated in our patient (*Figs.* 104–107) that there was no undue sacculation of the biliary tree and that emptying occurred promptly into the

duodenum, we removed the four-way tube by simply pulling it out. The fistula discharged bile for a few days, after which time it closed without any further

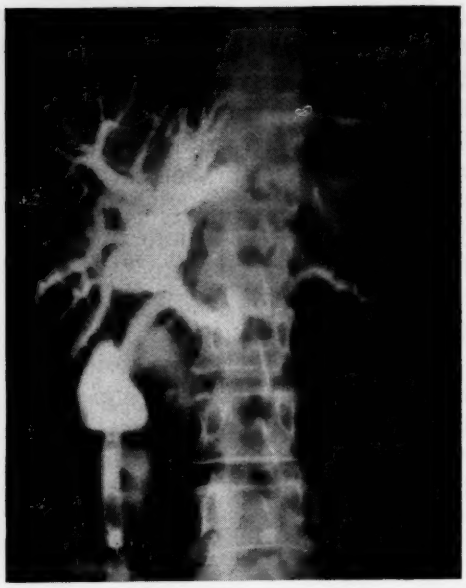


FIG. 104.—Injection of 40 c.c. of thorotrast through four-way tube into liver. (Tube should not yet be removed.) Note: (1) Clubbing and cylindrical dilatation of intrahepatic tree; (2) Thorotrast in duodenum proving patency of ampulla; (3) Thorotrast in pancreatic duct.

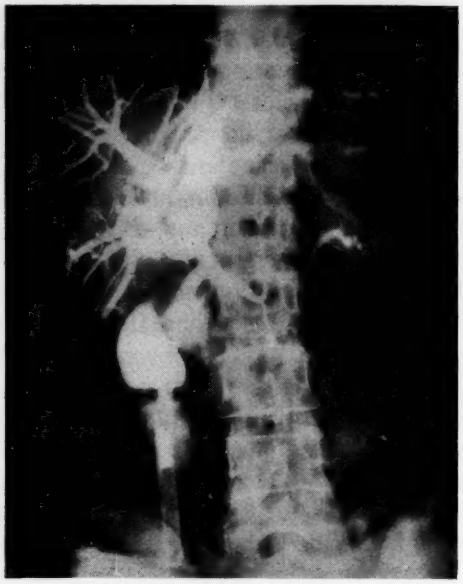


FIG. 105.—Film taken twenty minutes later than *Fig.* 104. Note: (1) Still some clubbing and dilatation of intrahepatic tree (2) Poor emptying.

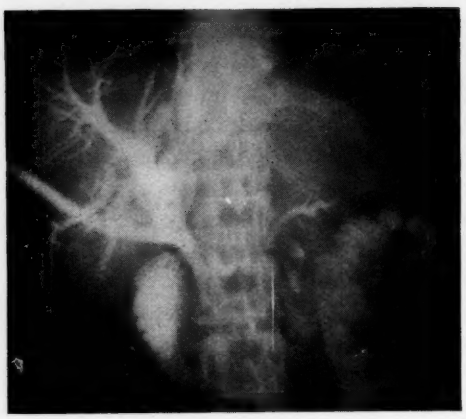


FIG. 106.—Injection of 40 c.c. of thorotrast through four-way tube into the biliary system. (Tube ready to be removed.) Note: (1) Disappearance of clubbing and cylindrical dilatation and more normal tree extending in fine ramifications to edge of liver; (2) Thorotrast in duodenum proving patency of ampulla; (3) Pancreatic duct.

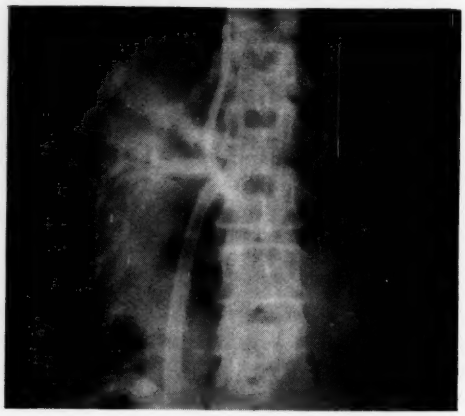


FIG. 107.—Film taken twenty minutes later than *Fig.* 106. Note: (1) Good emptying of biliary tree.

operative interference. Since that day, over three years ago, she has been in very satisfactory health, showing no jaundice, appetite good, still thin, occasional attacks of nausea and 'biliousness', but no pain, no fever, and no chills.

In conclusion we may mention one or two principles which seem to have been of importance in this case. Undoubtedly, the greatest single factor in the keeping out or getting out of trouble in any difficult surgical procedure is painstaking and adequate exposure. This will safeguard the tissues from mechanical damage, but in the case of the common duct, will not guarantee against later scar stricture nor prevent stricture arising from inflammation or that due to sloughing of part of the wall of the common duct, a consequence which may result from too enthusiastic dissection. If, during cholecystectomy, it is recognized that damage to the common duct occurs, it should be repaired at once, although this does not ensure against later stricture formation.[9]

The type of reconstruction chosen in this case and the means adopted for carrying it out were further factors contributing to the successful outcome. The four-way tubes, improvised and used as illustrated, ensured a free outlet for the bile from both right and left lobes of the liver to the duodenum, the additional limb to the surface being merely a safety-valve. The liver was decompressed and suffered no damage, and the patient was at all times receiving her usual quota of bile so that the digestion did not suffer.

In this particular case the post-operative transfusions were of inestimable value, for by them only was the patient kept alive. We are fully cognizant of the many elaborate tests to foretell hæmorrhage following operation on jaundiced patients, but in spite of all that have been described, there is still no way of predicting which jaundiced patient will bleed post-operatively. The safe rule is to assume that all will bleed and to treat them accordingly, before and after operation.

Furthermore, there is no sure preventive or cure for this most troublesome tendency. The normal and best stimulus for clotting is hæmorrhage, and in most cases oozing stops after a certain time regardless of vigorous or mild efforts on the part of the surgeon. The one and only therapeutic standby which weathers the test of time is whole blood transfusion. The addition of normal blood to the circulation provides something which the patient lacks, and by so doing stimulates clotting. Transfusions administered for this purpose rather than for the purpose of replacing blood-loss need not be large. Repeated small transfusions of 200 c.c. daily are better than fewer, larger ones.

Finally, the use of a T tube, or some such mechanical device about which to build the new duct, is necessary, and this tube must be left in position for many weeks. Its purpose is not merely to act as a mould about which the duct may be fitted, but rather to mould the growth and repair efforts of nature so that there may be a patent channel from both lobes of the liver to the duodenum. When one remembers that around such damaged tissues healing must occur by granulation and scar tissue, it becomes obvious that the supporting tube must remain in position until all fibrous tissue has been laid down, has contracted to its full extent, and has been held in check by the tube. The rigid fibrous channel thus formed must also be lined with epithelium so that the once patent channel may remain open and not heal across. It is not until this stage, which takes from six months to two years, has been reached, that the new duct is safe from further stricture by contracting scar or from obliteration by fusion of its walls, and therefore it is not until this time that the tube may be removed.

During the time that the reconstructed duct is reaching a state of secure equilibrium, a second equally important effect is being accomplished. The damaged

liver and intrahepatic biliary tree is decompressed by virtue of the ready egress provided for the bile, and thus is permitted to recover from any back pressure effects or inflammation. The dilated ducts contract and emptying is prompt, both evidences of good function. These changes may be watched and proven by X rays following injection of thorotrast into the biliary tree.

I wish to acknowledge the indebtedness of both the patient and myself to Dr. Roscoe R. Graham and Dr. James E. Barry, at the time of operation and also during the immediate post-operative difficulties.

REFERENCES

[1] *Exodus* v. 6–7.
[2] HORGAN, E., *Reconstruction of the Biliary Tract*, 1932. New York: MacMillan.
[3] COORS, GILES, "Reconstructive Surgery of the Common Bile-duct", *Southern Med. Jour.*, 1935, xxviii, 351.
[4] THORLAKSON, P. H. T., and MCMILLAN, J. C., "Common Duct Obstruction with Lipiodol Studies of Cholangiectasis and the Effects of Prolonged Drainage", *Canad. Med. Assoc. Jour.*, 1934, xxxi, 265.
[5] GABRIEL, W. B., "Proof of Patency of Common Bile-duct by Injection of Lipiodol", *Lancet*, 1930, i, 1014.
[6] OVERHOLT, R. H., "Biliary Tract Visualization with Radiopaque Oil", *Surg. Gynecol. and Obst.*, 1931, lii, 92.
[7] GINZBERG, L., and BENJAMIN, E. W., "Lipiodol Studies of Post-operative Biliary Fistulæ", *Ann. of Surg.*, 1930, xci, 233.
[8] JUDD, E. S., and PHILLIPS, J. R., "The Potency of the Biliary Ducts", *Surg. Gynecol. and Obst.*, 1933, lvii, 668.
[9] HUNT, VERNE C., "Reconstruction of the Common Duct", *Surg. Clin. of N. America*, 1934, xiv, 1389.

CONTINUOUS INTRAVENOUS INFUSION

By P. R. ALLISON

FROM THE DEPARTMENT OF SURGERY OF THE LEEDS UNIVERSITY AND THE GENERAL INFIRMARY AT LEEDS

It is now almost universally appreciated that the intravenous route for administering salines offers a rapid and sure method of increasing the body fluids and the salt content of the tissues, and that this may be a very important part of the treatment of shock, persistent vomiting, and toxic states. The method of continuous drip infusion has been used for many years, but little work has been done to determine the fate of the various substances which may be given in solution. The present work has been undertaken for the purpose of estimating the value of some of these methods. The clinical and biochemical observations made on a series of cases will be published later. This communication is concerned with a description of the apparatus that has been devised and which is now in use in the wards at the General Infirmary at Leeds.

Although from the clinical point of view it is satisfactory to give saline for short periods and at a very slow rate without regard to the temperature of the fluid entering the vein, the addition of a heating mechanism has the very marked advantage that the rate of flow of the fluid can be regulated within wide limits and that in an emergency one or two pints can be introduced into the circulation in ten minutes or a quarter-of-an-hour. In the General Infirmary at Leeds the standard method of giving intravenous salines during the last few years has been by means of a suspended thermos flask and a Murphy's drip. The disadvantages of this method are manifold. The thermos flask can never be adequately sterilized; it has to be changed frequently owing to its small capacity, the apparatus must be dismantled for replenishing, during which time the blood enters the cannula and clots; and there is nothing to call the attention of the nurse to the fact that the flask is nearly empty. The greatest objection to the apparatus, however, is that it fails to achieve the end for which it is specifically designed—that is, to deliver warm saline into the vein. It is not sufficiently realized that the fall of temperature along a rubber tube in such an apparatus is extremely rapid, and that the passage of the fluid through only a few feet of the tube is sufficient to reduce its temperature to that of the room.

Two of the main difficulties have been overcome in the following way: a large reservoir made of glass and holding enough saline for twenty-four hours was graduated so that the volume of its contents could be read off at any time, and the fluid was heated to the required temperature close to the point at which it entered the vein by means of a thermo-regulated water-bath. The temperature of the bath could be adjusted within fairly wide limits. In the early experiments the fluid was passed through a glass spiral immersed in the bath with the result that gases were liberated and air-locking occurred. The spiral was therefore replaced by the glass apparatus shown in *Fig.* 108. The fluid passes through a side tube C into

a wide vertical tube A, where it is heated to the temperature of the bath. From the lower end of this it passes into an outflow tube B, which is of narrow bore, and so on through a length of pressure tubing and a cannula to the patient's vein. As the limb of the tube A is of relatively large capacity, the rate of flow through this part of the apparatus is slow and the fluid is heated to the temperature of the bath before it reaches the limb B. The gases are therefore liberated in the wide limb A and provision is made for their collection in a bulb D above. The gas as it accumulates can be removed at intervals of twenty-four hours through an outlet tube E in the top of D. This has been called an 'infusion air trap', and it is made by Messrs. Reynolds & Branson Ltd., of Leeds. It fits firmly into a cork which holds it in a hole in the top of the water-bath. The dimensions are as follows: the wide limb is 1 in. in external diameter and 6 in. long from the bulb downwards. The narrow limb is $\frac{1}{4}$ in. in external diameter and 6 in. long, and the bulb itself is 2 in. in diameter. In order to increase its strength a metal ring encircles the two limbs of the tube, and at this point there is wedged between them a small piece of cork. When the trap is in position the inlet C is separated from the hot lid of the water-bath by a sheet of asbestos so that no warming of the fluid takes place until it reaches the limb A of the air trap. Before this precaution was taken air was liberated in the tube between the drip and the trap, and locking occurred. It is very simple to estimate the rate of fall of temperature along that part of the tubing beween the source of heat and the patient for different rates of flow and for different temperatures of the bath with a constant room temperature. This is done by piercing the wall of the rubber tube at intervals and inserting thermometers along its length. The rate of flow is conveniently plotted as drops a minute in the Murphy's drip. This information provides the means whereby the temperature of the fluid as it enters the patient's vein can be estimated within a few degrees in widely varying circumstances.

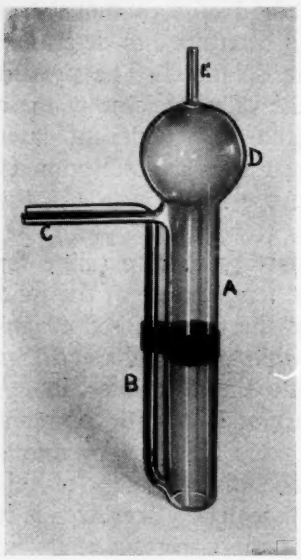


FIG. 108.—The infusion air trap.

Fig. 109 shows the method in use at the General Infirmary at Leeds, where the investigations have been carried out during the last two years. The infusion fluid is held in a large graduated flask which has a capacity of eight pints, a metal lid which can easily be removed, and a funnel-like outlet below. This is sterilized and hung on a movable metal stand at the bedside. A Murphy's drip is attached to the outlet by a short length of rubber tubing, all of which are sterilized by boiling. The flask is then filled with the required solution. From the drip a rubber tube passes to the air trap and the whole system is filled with fluid to remove air. Pressure tubing bearing the glass cannula is attached to the exit from the trap. On this piece of tubing is a metal clip so that the system can be cut off from the circulation when adjustments are necessary, as for example the removal of air from the glass bulb. When the apparatus is once assembled it has the great advantage that no attention is required apart from the daily refilling of the flask, and as this does not necessitate any interruption of the flow it is a thing which can be done by any

nurse who has even an elementary knowledge of asepsis. When salines are to be infused for some days a glass cannula tied into a vein is more satisfactory than a needle, and I am indebted to Mr. R. A. Hall for his suggested use of the cephalic vein in the middle of the upper arm. A cannula tied into this vein and covered with adhesive strapping allows almost full use of the arm and hand.

The thermostatic bath was made by Falk Stadelmann & Co. Ltd., of Leeds. It is a copper bath 12 in. in each dimension, and the heat supply is taken from the electric mains. It is fitted into a special table of which two only of the legs are provided with wheels so that when at rest it is stable, and yet when one end is lifted

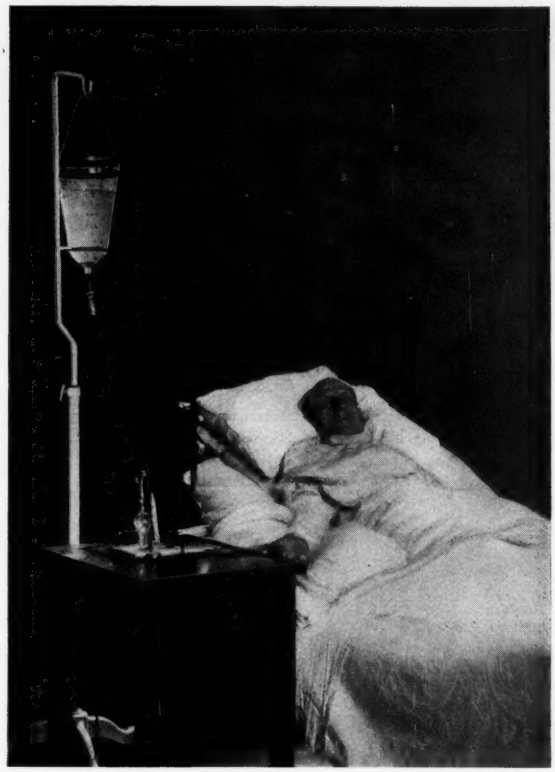


FIG. 109.—Patient receiving continuous intravenous infusion. An early case before the use of the cephalic vein was made a routine.

it is easily moved about on the two wheels. When the infusion is once started this apparatus will continue to deliver the fluid to the patient at a known temperature and rate so long as the reservoir is replenished at intervals and so long as the vein remains patent. Owing to the constant and uninterrupted pressure of fluid no reflux of blood into the cannula, with consequent clotting, occurs. After some days, however, the irritation of some fluids causes an aseptic phlebitis with swelling of the vein wall and gradual occlusion of its lumen. When this occurs it is a simple matter to fix the clip on the delivery tube in order to cut off the flow and then to change the cannula and fix it into another vein, usually the corresponding one on the other arm.

The total cost of the whole apparatus can be greatly reduced by replacing the thermostatic bath by a small wooden box containing one or two electric light bulbs and a thermometer. A hole is made in the roof of the box and into this the cork bearing the air trap is fitted. It is, of course, not possible to regulate the temperature as easily as with the more expensive apparatus, and it therefore requires more careful attention.

The following is a brief summary of a typical case history to indicate the value of this form of treatment. Only those details which are relevant to this communication are given, but they are sufficient to indicate the benefits which can be expected and the lines along which the investigations are being conducted.

F. W., male, aged 59 years, was admitted to the General Infirmary at Leeds under the care of Mr. Flint, on Sept. 26, 1934. with a history of fourteen years' epigastric pain half an hour after food, together with flatulence and heartburn. Three years after his first attack a duodenal ulcer had perforated, and following suture of the ulcer he had had relief from symptoms for one year. Since that time there had been repeated attacks of pain of less severity with a recent complaint of vomiting. He was a very emaciated subject for his build, his stomach was distended down to the umbilicus, and he was a typical example of neglected pyloric stenosis. He was treated in bed and given daily stomach washes and continuous intravenous glucose saline infusion for a period of eight days, after which a posterior gastro-enterostomy was performed. During the preliminary treatment his stomach contracted notably, and his general condition improved beyond all recognition, as the following table shows :—

Day	Urine	Intravenous Saline	Weight
	Oz.	Pints	
1	8	nil	7 st. 10 lb.
2	16	4	
3	34	4	
4	52	5½	
5	92	6	
6	110	6½	
7	88	3½	
8	88	6	
9	140	3½	8 st. 7½ lb.

At no time during the treatment did any sugar appear in the urine. The total gain in weight was 11½ lb., due almost entirely to the fluid which had been retained in the tissues. The strength of the glucose solution was 6 per cent, so that the total quantity of glucose metabolized was about 110 grm. a day, taking the average rate of infusion as four pints in the twenty-four hours.

TWO CASES OF GANGLIA IN THE SHEATH OF THE PERONEAL NERVE

By V. H. ELLIS

ORTHOPÆDIC SURGEON, ST. MARY'S HOSPITAL, LONDON

Cystic degeneration of a nerve-sheath is a condition which does not appear to be mentioned in text-books of pathology or neurology, but several cases have been recorded in Continental, though none has been found in the English, literature. This condition is associated with sensory changes and paralysis of the affected nerve, and it is interesting that out of 13 recorded cases, the peroneal nerve has been involved on 7 occasions.

The earliest case appears to have been reported by Hartwell,[1] of Boston, in 1901. There was a cyst of the size of a hazel-nut in the median nerve with impaired mobility of the forearm and loss of sensibility to pain and touch. On operation the cyst was found to contain a watery and mucoid substance. Complete recovery followed its evacuation. The writer knows of one somewhat similar case where the median nerve was also involved.

Cysts of the peroneal nerve have been recorded by Sultan,[2] of Kiel, Zaar,[3] and Wadstein.[4]

Etiology.—The frequency with which the peroneal nerve is involved as it winds round the neck of the fibula suggests that trauma plays a part in the causation of the condition, as perhaps does its analogy to the very similar ganglia on tendon-sheaths. Zaar lays emphasis on the great part played by trauma; nevertheless a definite history of injury is not always obtainable.

Symptoms.—In nearly all cases the first symptoms are those of pain in the areas supplied by the affected nerve. The pain varies in intensity and may be accompanied by numbness; after a few weeks the patient notices weakness of the extensor muscles and complains of drop-foot.

Diagnosis.—The patient's chief complaint is usually that of foot-drop. On examination of the leg a swelling can be felt in the course of the peroneal nerve which may be tender. It is cystic in nature, may be lobulated, and tender to touch, extending from the front of the neck of the fibula some distance up the nerve behind the biceps tendon. The swelling is not attached to skin or deeper structures; it is never associated with swellings on other nerves or signs of a generalized neuro-fibromatosis.

Pathology.—The tumour forms a fusiform translucent swelling lying under the sheath of the nerve-trunk and occasionally following one or more of its branches into the muscles of the leg. The tumour is lobulated and lies only partially between the nerve bundles. The wall of the cyst is composed of connective tissue without nervous elements. The contents are a colourless, glairy, jelly-like fluid, exactly similar to that found in ordinary ganglia.

Treatment.—Simple incision of the cyst and expression of its contents can be performed without injury to the nerve. The skin is sutured and heals soundly.

There appears to be no liability to recurrence. In cases of short duration the sensory symptoms disappear early and the paralysis passes in the course of a few months.

CASE REPORTS

Case 1.—Stanley C., aged 13. First seen on Dec. 3, 1929, complaining of pain in the dorsum of the left foot and drop-foot of one month's duration. There was a tender swelling ½ × 1 in. just below the head of the fibula. An X-ray showed no bony changes.

OPERATION (Jan. 17, 1930).—The nerve proximal to the head of the fibula was twice as thick as normal, looked œdematous, and felt hard; there were several small translucent cysts. Below the head of the fibula the peroneal branches were intact, and the muscles responded briskly to electrical stimulation. The anterior tibial nerve was apparently replaced by a swelling as large as a walnut, consisting of many freely communicating cystic cavities. This cyst was incised and evacuated. The cysts higher up were milked down and expressed through the same opening. The incision was then closed.

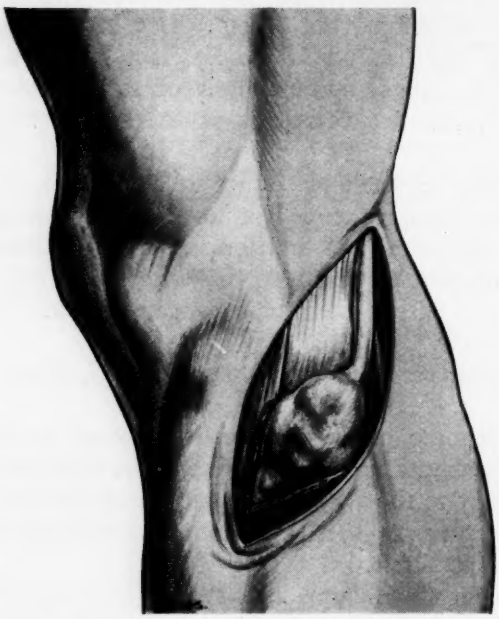


FIG. 110.—*Case* 2. View of the outer side of the left knee showing exposure of peroneal nerve with the ganglion lying below the head of the fibula partially in the substance of the peroneus longus, which has been cut.

Aug. 5: Electrical reactions: peronei, normal; anterior tibial group, weak response; tibialis anticus did not appear to respond.

Dec. 2: Complete recovery.

Case 2.—John J., aged 44. A corn warehouseman.

Oct. 14, 1935: History of moderate pain in the left foot and foot-drop for one month. There was no definite history of injury, but his work involved filling heavy sacks of grain which he held between his knees, often resting the outer side of his left knee against another sack for support.

ON EXAMINATION.—Paralysis of muscles supplied by anterior tibial nerve. Over the head of the fibula, on its lateral aspect, there was a cystic swelling of the size of a cherry. The swelling was smooth and rounded, not tender, and doubtfully translucent; not attached to skin or bone, and could be moved slightly from side to side. All the muscles of the anterior group reacted normally to faradism and galvanism.

OPERATION (Oct. 17).—Exploration of left external popliteal nerve (*Fig.* 110). Thin-walled cyst found in sheath of nerve extending for a short distance into peroneus longus muscle. Nerve-fibres compressed but intact. The cyst contained a jelly-like fluid.

Histological Report on Cyst Wall.—Dense fibrous tissue with a lining of flattened cells. No nervous tissue.

Oct. 30: The patient was discharged with a toe-raising spring fitted to the boot.

Jan. 12, 1936: The paralysis had diminished after five weeks, and the function of the foot was normal by this date.

I am indebted to Mr. S. L. Higgs for permission to publish *Case* 1, which was under his care.

REFERENCES

[1] HARTWELL, *Med. and Surg. Jour.*, 1901, No. 24.
[2] SULTAN, C., *Zentralb. f. Chir.*, 1921, No. 27.
[3] ZAAR, *Ibid.*, 1926, 2551.
[4] WADSTEIN, T., *Acta Orthop. Scand.*, 1931, ii, 221.

CYSTIC DISEASE IN SUPERNUMERARY BREASTS

By A. J. NORONHA

LECTURER IN PATHOLOGY AND BACTERIOLOGY, B.J. MEDICAL SCHOOL, POONA

In 1883 Reclus separated a disease which he called 'maladie kystique de la mamelle'. Since that time the affection has been described under a variety of names—for example: 'maladie de Reclus', 'Schimmelbusch's disease', 'chronic mastitis', 'chronic cystic mastitis', 'cystic disease of the breast', 'chronic interstitial mastitis', and 'cystadenoma papilliferum'.

The following case was referred to me for opinion by Mr. H. S. Mehta, M.S., Civil Surgeon, Dhulia, Kandesh, India.

"Mrs. S., was admitted for swelling in both the axillæ. She is a multipara, the last confinement being her third. She is 24 years old. She states that the swellings above referred to were noticed on the 'first day of her last pregnancy',

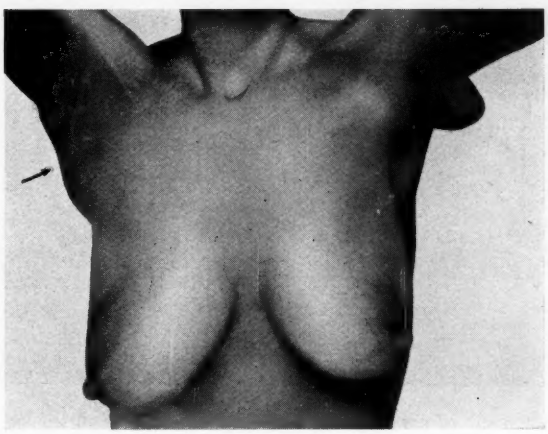


FIG. 111.—Photograph of patient, showing the two axillary tumours, one on either side.

namely, about a year prior to her admission into the hospital. The swellings increased progressively to their present dimensions. After her delivery she noticed that these swellings used to diminish in size whenever she nursed her child. On examination, both the tumours were found to be soft, fluctuating, freely movable, specially the one in the left axilla. The latter appeared to have no connection with the left breast. Aspiration of both swellings brought out milk. The tumour on the left, being more easily removable, was enucleated and sent for examination." So far Mr. Mehta's notes.

On the evidence to be shortly described, I diagnosed the tumour as cystic disease of a supernumerary breast. Mr. Mehta very kindly supplied me with some photographs along with the excised material. One of these is reproduced

in the text (*Fig.* 111). On examining the sections I requested Mr. Mehta to excise the second tumour and to send it for examination. This request was readily complied with, and about a month later the second tumour arrived with the statement that it appeared to be connected with the right breast by a fascial attachment and that no actual communication with this organ had been discovered. Moreover, no oozing of the contents had occurred on removal.

I cut open each tumour on arrival and collected the contained fluid in test-tubes. In each instance the material had a rancid odour, similar to that of stale condensed milk. The colour was yellowish-white and the consistency that of inspissated milk. Curdy material was present in certain parts of the tumour.

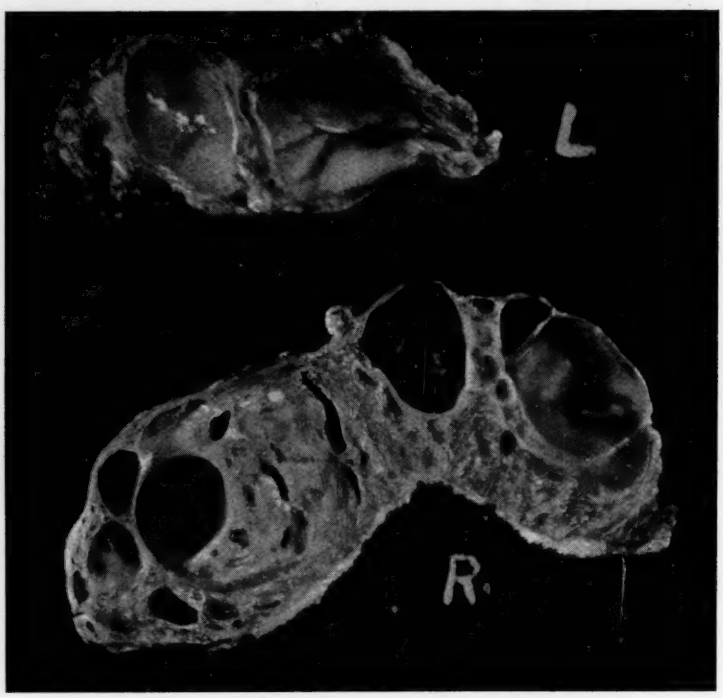


FIG. 112.—Photograph of gross appearance of the tumours submitted for examination.

It cannot be ascertained with any confidence that this was the result of the formol-saline used in fixation, though this does not appear improbable. A drop of the liquid was examined under the microscope with a low power. It was found to consist of fat globules, similar to those seen in ordinary milk. Application of Benedict's test showed the presence of a reducing substance, and a positive test for protein was obtained with Bogg's reagent.

The naked-eye appearance of the two tumours is shown in *Fig.* 112. The illustration ($\frac{3}{4}$ size) shows their exact shape, except for a nipple-like projection in the larger tumour, caused by retraction of the overlying skin. The left tumour is the smaller of the two and is marked L. The right (marked R) is the larger. Both have been enucleated in their entirety and had not been cut into on removal. In the description that follows, the two tumours will be referred to as Tumour L and Tumour R respectively.

Tumour L shows two large cavities on section and many small ones, being honeycombed towards the edges and on the partition between the larger cysts. Sections from the honeycombed areas show lactating acini. In the photomicrograph illustrating these (*Fig.* 113) one can see a dilated duct with papillary processes projecting into the lumen.

Tumour R, as seen in *Fig.* 112, is the larger of the two. The cystic dilatations are more numerous and of variable size. In the left half near the middle line, the honeycombed area is filled with a white curdy material. Sections from the less affected areas show lactating acini, but there is greater profusion of dilated ducts, some of which show proliferation of the lining epithelium (quite apart from the effect of oblique section). In some places a well-marked round-cell infiltration is present. In other sections, larger papillomata, to all appearances, severed during cutting are present within the lumen of dilated ducts, while in other places acidophil cells and epithelial debris are present within the lumen of the ducts.

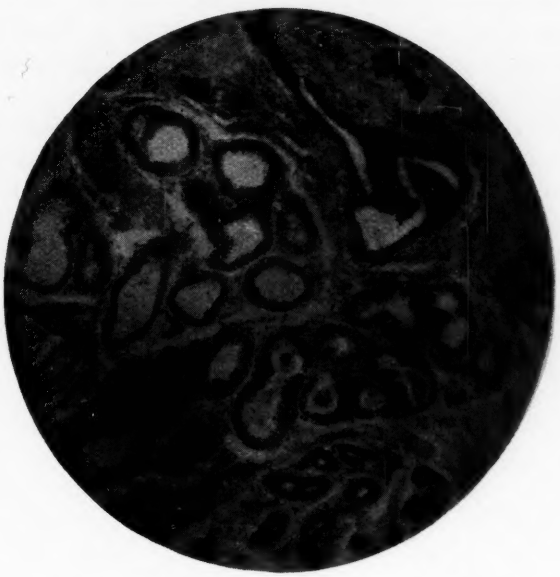


FIG. 113.—Photomicrograph of tumour of left side.

The case is published because a bilateral affection of supernumerary breasts is present in the patient. The interest is further enhanced by the fact that the removed breast tissue does not, as far as I could ascertain, communicate with the adjacent breasts. Moreover, there is no external communication nor even a rudimentary nipple.

The pathology of cystic mastitis is a matter of dispute, and there is great difference of opinion as to its causation. In France it would appear to be common in young women (Letulle), while other authorities hold the view that it occurs on the wrong side of middle age. Keynes's statistics apparently show that in England the disease occurs more frequently in "women nearing the menopause"—that is, between 30 and 40 years.

The theories as to the pathogenesis of this affection are as numerous as the names by which it is known. To mention only a few, it is held by some that the

condition is inflammatory. MacFarland, after an analysis of numerous cases, upholds Warren's theory of " abnormal involution ", or " delayed involution " as MacFarland puts it. Schimmelbusch adheres to the neoplastic theory. In his Hunterian Lecture, delivered before the Royal College of Surgeons in England on May 9, 1923, Geoffrey Keynes arrives at the following conclusions, among others :—

" 1. The breast is a secretory gland which shows periodical activity from birth to menopause.

" 2. The normal non-lactating breast has no outlet through the nipple for the discharge of its secretions. Secretion must therefore be balanced normally by reabsorption.

" 3. Chronic mastitis is manifested by dilatation of ducts and acini, accumulation in them of the products of epithelial activity, infiltration with lymphocytes, fibrosis, and epithelial changes. Distribution of all these is very erratic.

" 5. Chronic mastitis is not bacterial in origin, toxæmic, or traumatic. Nor is it related to involution changes in the breast.

" 6. The cause of chronic mastitis is probably to be found in chemical irritation due to stagnating secretions and epithelial debris. This cannot be proved until the chemical changes have been investigated."

I mention these conclusions in detail, as the case above described would appear, in a measure, to substantiate Keynes's contention. In the first place, the affection is that of supernumerary breasts without any outlet for the resultant secretions. Secondly, the trouble arose during lactation, though one could not ascertain whether any hypertrophy of the additional breast tissue occurred during previous pregnancies. It would appear from the history that the patient did not notice anything previously. Still, it is well known that during lactation some acini may remain dormant. The theory of stagnating secretions would appear to apply particularly to the case under consideration.

BIBLIOGRAPHY

BOYD, *Surgical Pathology*.
KEYNES, *Brit. Jour. Surg.*, 1923, xi, 89.
LETULLE, MAURICE, *Anatomie pathologique*.
MACFARLAND, *Surgical Pathology*.

THE SIGNIFICANCE OF LUMBOSACRAL TRANSITIONAL VERTEBRÆ

By G. A. G. MITCHELL,
SURGICAL SPECIALIST FOR THE COUNTY OF CAITHNESS

MAN'S assumption of the erect posture has necessitated many skeletal modifications and these are nowhere more apparent than about the lumbosacral junction. Finality in structure has not yet been attained and developmental abnormalities are abundant, a fact of practical importance, as some anomalies strengthen the spine while others weaken it. The presence of an enlarged transverse process on one or both sides of the last lumbar vertebra is one of the best known of these anomalies, and it is interesting both from the anatomical and clinical view-points. The condition is usually referred to as sacralization or lumbarization, and its occurrence is puzzling unless one possesses a knowledge of the anatomy and morphology of the spinal column.

ANATOMICAL SIGNIFICANCE

The normal formula for the human vertebral column is: C.7, T.12, L.5, S.5, Co.4, but variations from this occur in about 20 per cent of skeletons. The total number of segments seldom varies, however, and as there is no proof that intercalation or excalation of whole segments occurs naturally, any addition of a vertebra to one region must be associated with the loss of a vertebra from another. Numerical variability is greatest at the sacro-coccygeal junction, and usually results in the acquisition of an extra vertebra by the sacral region and a loss of one from the coccygeal region, but variations at the lumbosacral junction are also common, resulting in either elongation or shortening of the lumbar region.

Phylogenetic advances have been accompanied by reduction of segments at both the cranial and caudal ends of the spinal column. In the evolution of the higher primates the lumbar region has become relatively shortened, the process of reduction being brought about by the transformation of lumbar into sacral vertebræ. As a consequence the whole pelvic girdle has migrated in a cranial direction and the number of pre-sacral vertebræ has decreased. According to Rosenberg these phylogenetic advances are partly recapitulated in human ontogeny, because the pelvic girdle is primarily attached to the twenty-sixth vertebra, but subsequently extends forwards on to the twenty-fifth, while at the same time the thirtieth vertebra, primarily sacral, becomes separated from the sacrum to form the first coccygeal vertebra. Rosenberg's conclusions are not accepted by all authorities; indeed the balance of opinion seems to swing more to the opposing view that in man the pelvis is migrating backwards instead of forwards. If the latter opinion is correct, Rosenberg's theory must be incorrect or incomplete. Most anatomists are agreed that it is correct but incomplete, and to complete the theory Keith has suggested that in the human phylum the lumbar region was again lengthened when the plantigrade mode of progression was substituted for the brachiating.

An idea of the tendency to progression or retrogression of the pelvis can be gained by studying the number of pre-sacral vertebræ. The number of cervical vertebræ remains practically constant, and thus any increase or decrease in the pre-sacral vertebræ usually affects the thoracico-lumbar region. The normal formula for these regions in man is T.12, L.5, giving a modal number of seventeen. If the pelvic girdle progresses in a cranial direction, a lumbar vertebra becomes incorporated in the sacrum and the thoracico-lumbar segments are reduced to sixteen, whereas if it recedes caudally the process is reversed and the number of thoracico-lumbar vertebræ is increased to eighteen. The spinal column of man shows less tendency to numerical variations than that of anthropoid apes, and is characterized by the stability of the modal number of seventeen. Todd has observed that the modal number of the gorilla and chimpanzee is also seventeen, of the gibbon eighteen, and of the ourang sixteen. Thus at first sight it appears that the ourang has progressed further than man, but if Keith's theory is correct it has not this significance, since he suggests that man has reached and passed this stage, and that his lumbar region is becoming elongated again. Rosenberg, Todd, and Willis think that in man the tendency is towards reduction in the number of the thoracico-lumbar segments to sixteen. On the other hand Bardeen, Frets, Keith, Dwight, Fischel, and Shore are of the opinion that the pelvis is migrating backwards in man, tending to increase the number of thoracico-lumbar segments to eighteen. If the almost constant cervical element of seven is added, it is clear that the total number of pre-sacral vertebræ oscillates between twenty-three and twenty-five, and the numerical variations are mainly dependent on a reciprocal gain or loss of one vertebra at the lumbosacral junction. Often the process is incomplete and the vertebra gained by the sacral from the lumbar region, or vice versa, fails to assume completely all the characters of its adopted region, and this explains why transitional types of vertebræ are common at the lumbosacral junction. The transitional vertebra may have lumbar characteristics on one side and sacral on the other, and it is either the twenty-fourth or the twenty-fifth of the pre-sacral series. Clinically this condition is usually referred to as ' sacralization of the fifth lumbar vertebra', but if Keith and other observers who maintain that the pelvis in man shows a preponderating tendency to migrate caudally are correct, then the condition is more often a lumbarization of the first sacral than a sacralization of the fifth lumbar vertebra. The tendency for the sacrum to acquire a segment from the coccyx is greater than its tendency to lose one to the lumbar region, and Shore suggests that in the evolutionary sense acquisition of the thirtieth vertebra (normally the first coccygeal) by the sacrum precedes, and perhaps even makes possible, the release of the twenty-fifth vertebra (normally the first sacral) to the lumbar region. By simply looking at an isolated specimen or a radiograph of the lower spine it is impossible to tell whether sacralization or lumbarization is present; the total number of pre-sacral vertebræ must be known before an opinion can be given.

The most striking peculiarity of the transitional vertebra is the appearance of one or both transverse processes, which are greatly enlarged as compared with the normal lumbar type (*Fig.* 114). These processes are really a combination of transverse and costal elements, the costal being the larger. In a normal first sacral vertebra the anterior two-thirds of the ala sacralis represents the costal and the posterior third the transverse process (*Fig.* 115). Even in a normal fifth lumbar

vertebra (*Fig.* 115) the costal elements form a not inconsiderable component of the transverse processes, but when sacralization is present they have undergone a degree of development analogous to that which normally occurs in the upper sacral vertebræ.

The abnormal costo-transverse processes may be large or small, symmetrical or asymmetrical. They may not be enlarged sufficiently to reach either the sacrum or the ilium, but at other times they articulate with the sacrum (*Figs.* 114, 116), or even with the ilium, on one or both sides. Sometimes there is unilateral or bilateral bony union between the costo-transverse processes and the sacrum (*Figs.* 117, 118), though the processes are often asymmetrical, and the vertebral bodies and arches

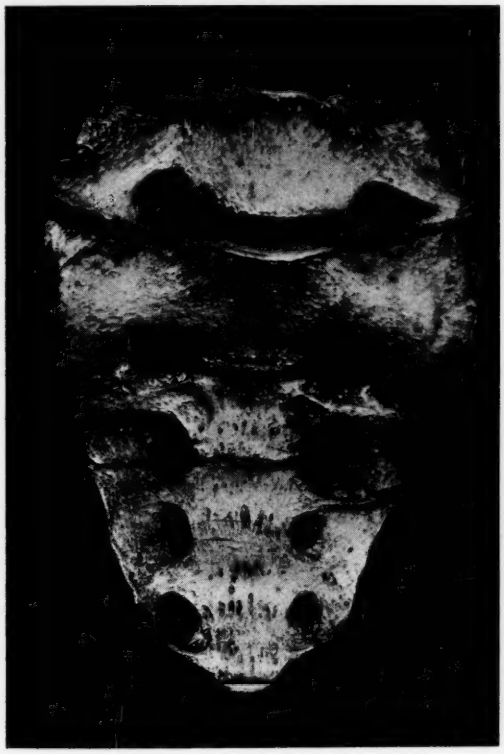


FIG. 114.—A lumbosacral transitional vertebra with bilateral costo-transverse articulations. It is impossible to tell whether this is an example of lumbarization or of sacralization as the rest of the vertebral column was not available for examination.

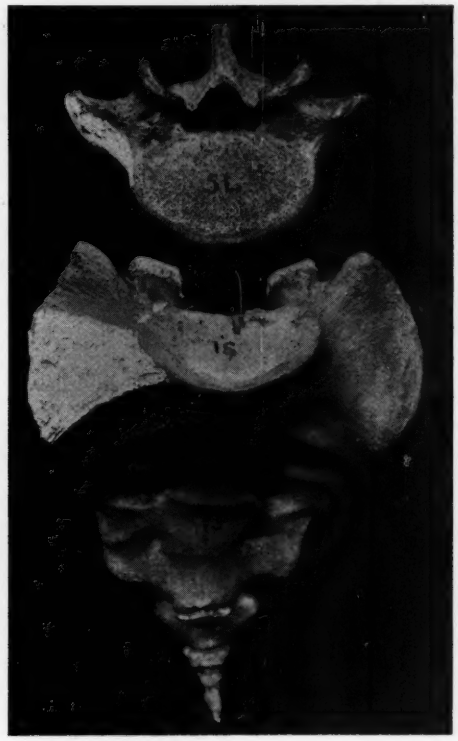


FIG. 115.—The whitened portions indicate the costal elements in the last lumbar and first sacral vertebræ.

may remain partly or completely unfused. Absolute symmetry is uncommon. In about 50 per cent the transitional vertebra shows lumbar characteristics on one side and sacral on the other (*Fig.* 117), the remainder showing anomalous developments on both sides (*Fig.* 114). The body of the transitional vertebra is usually wedge-shaped from side to side, being wider on the side with the larger costo-transverse process. The sacral auricular surface may encroach on the transitional vertebra (*Fig.* 119). If the twenty-fourth vertebra (normally the fifth lumbar) becomes united to the sacrum by bilateral bony union, the process of sacralization

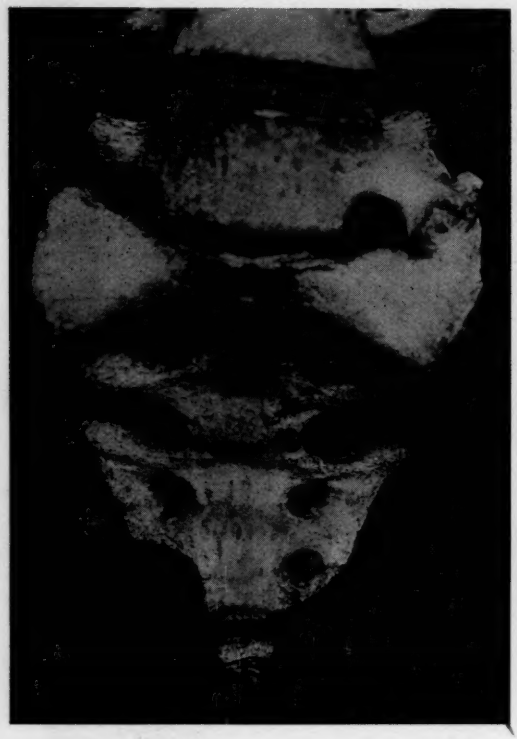


FIG. 116.—Unilateral incomplete lumbarization.

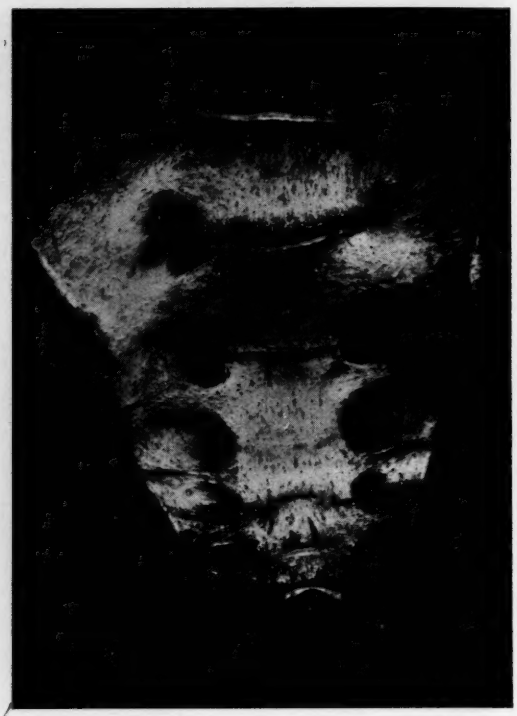


FIG. 117.—Unilateral complete sacralization.

FIG. 118.—A six-piece sacrum.

is said to be complete. This may result in a six-piece sacrum (*Fig.* 118), unless a segment is acquired by the coccyx from the sacrum, thus maintaining a five-piece sacrum. Lumbarization, by partially or completely releasing the first sacral vertebra to the lumbar region, would produce a four-piece sacrum were it not for the fact that the five segments of the sacrum are usually maintained by the acquisition of the first coccygeal segment. Four-piece sacra are rare, only occurring in about 1·2 per cent of European skeletons (Le Double).

The frequency with which lumbosacral transitional vertebræ are to be expected varies in different races. Wetzel found that they were present in 18 per cent of Australian aboriginals; Shore estimated the percentage in Bantu natives to be 4·8;

FIG. 119.—Examples of encroachment of the sacral auricular surfaces on lumbosacral transitional vertebræ.

Toyoda discovered that they were present in 5·8 per cent of Japenese spines; Brailsford, from radiographic studies, gives the percentage as 8·1 for natives of Britain; and Keith states that "in a series of one hundred human skeletons it will be found that some three or four will show a headward sacralization, while six or eight show a tailward movement."

CLINICAL SIGNIFICANCE

Should complete sacralization of a fifth lumbar vertebra have occurred, the lumbosacral junction is strengthened, and this is true even if the bony union is present only on one side. Complete lumbarization of the first sacral vertebra

lengthens the lumbar portion of the spine, thus allowing slightly increased mobility, but as mobility and strength are opposing virtues the lumbosacral junction is weakened. If two well-formed lumbosacral transverse articulations are present the junction is strengthened, but trouble may arise from the liability of the abnormal joints to arthritic changes. In the other incomplete types there is little doubt that the enlarged costo-transverse processes are occasionally the cause of low back pain and other symptoms. Jones and Lovett say they are rarely responsible for symptoms, and Verrall agrees, but says symptoms are severe when they do occur. Léri says they seldom cause pain, but Mennell in this country and the majority of American writers believe that they are capable of causing symptoms. A few deny that they ever cause symptoms and attempt to justify their contention as as follows :

1. The anomaly has been present from birth, yet no symptoms may develop till adult life.

2. The condition may be found during routine examination in patients with no backache.

3. Many who have been operated upon and had this ' cause ' of backache removed have not been relieved.

These contentions are easy to refute :—

1. The process at birth is relatively small and partly cartilaginous, and does not reach its full development or become completely ossified till early adult life. Obviously a small, partly cartilaginous process is less liable to cause trouble than a larger, completely ossified structure.

2. Other congenital anomalies, e.g., cervical ribs and spina bifida, may be found by chance during a routine examination for some other condition, yet no one doubts that these anomalies occasionally produce symptoms.

3. It is true that the removal of an enlarged costo-transverse process does not always cure low back pain ; but that only proves that the diagnosis and treatment were wrong in that particular instance. The fact that appendicectomy after a misdiagnosis of appendicitis does not cure the patient is no reason for denying that the appendix ever causes pain.

There is a sufficient body of evidence to prove that these enlarged processes are potential and sometimes actual causes of low back pain. They may produce symptoms in various ways :—

1. If the process is not large enough to articulate with the sacrum, lateral bending movements may cause it to impinge on the sacrum, thus nipping the intervening soft tissues and bruising the periosteum.

2. In unilateral cases the impingement of the process on the sacrum or ilium may provide a fulcrum for the spinal column, which then acts as a lever of the second class tending to prise apart the lumbosacral transitional vertebra from the sacrum, straining or tearing the connecting ligaments (*Fig.* 120). It is obvious that the ligaments on the side opposite to the abnormal process will suffer most, and this explains the seeming paradox that the symptoms and signs are sometimes worse on the side with the normal process. Violent lateral bending may cause acute lumbosacral strain, while ordinary lateral bending movements may produce minor repeated ligamentous strains which have a cumulative effect. This type of strain is distinguished by certain writers from other cases of lumbosacral strain by calling it ' lumbosacral transverse strain,' but there is no advantage in this, for it is highly improbable that the ligamentous fibres surrounding the abnormal costo-transverse articulation suffer alone.

3. An adventitious bursa may form between the enlarged process and the sacrum or ilium and become inflamed owing to repeated small injuries.

4. If the process articulates with the sacrum or ilium, a so-called lumbosacral transverse articulation is formed. The articular cartilages of the joint are often thin and therefore not very efficient as shock absorbers, thus lessening the resistance of the joint to traumatic insults. Repeated minor injuries such as are produced by lateral bending, heavy lifts, etc., initiate arthritic changes more readily under these circumstances.

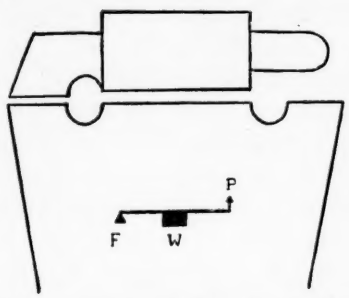


FIG. 120.—A diagram to illustrate how levering effects may be produced by unilateral incomplete lumbarization or sacralization.

5. In some cases associated peri-arthritic changes may involve adjacent nerves. Ecchondroses or osteophytes may develop around the margins of the abnormal articulations, encroach on the intervertebral foramina, and press on the contained nerves. In sacralization the fifth lumbar nerve is liable to be involved, while in lumbarization the first sacral nerve is the one liable to suffer.

6. Bauman thinks that the part of the fourth lumbar nerve which helps to form the lumbosacral trunk may be stretched as it passes over the enlarged costo-transverse process, particularly during spinal extension, and that this may give rise to radiating nerve pains.

SYMPTOMS AND SIGNS

Symptoms.—Symptoms due to lumbarization or sacralization often manifest themselves during the third decade, and in this respect the clinical history resembles that of many cases with cervical ribs; this is interesting when one considers that the symptom-producing costo-transverse process is mainly formed by an abnormal development of the costal element. The symptoms may first appear after injury (particularly a fall on the flat of the back), illness, unaccustomed activity, or working in a cramped or unusual position, while anything upsetting the normal posture or gait and throwing more strain on the side with the abnormal process, may initiate the trouble. One patient, for example, developed low back pain due to an incomplete right unilateral lumbarization after injuring a cartilage in his left knee, which led to recurrent attacks of synovitis and caused him to throw more weight on the side with the lumbarization. Verrall remarks on the intermittency of the symptoms; this may be due to recurrent inflammation of an adventitious bursa, recurrent arthritis in a lumbosacral transverse joint, or occasional nipping of tissues between the sacrum and the process.

Low back pain is the most common complaint, and it is fairly well localized over the lumbosacral region. In some cases it is very severe and may completely incapacitate the individual. In others it takes the form of an indefinite dull ache. Like most other forms of low back pain it is extremely intractable at times, and arouses the usual suspicion of neurosis or malingering. Lower spinal movements, walking, coughing, and abdominal straining increase the pain, while lifting weights and bending become difficult or impossible. Owing to the possibility of involvement of the lower lumbar or first sacral nerves, pains in their cutaneous areas of

distribution are not rare, occurring along the outer side of the leg and in the foot. Pains along the posterior aspect of the thigh are less common. In cases with definite nerve irritation or compression the patient may complain of weakness or unsteadiness of the leg on the affected side. Any nerve symptoms and signs in sacralization are practically identical with those which may occur in lumbosacral strain, whereas in lumbarization any nerve features present resemble more closely those which may occur in sacro-iliac strain.

Children rarely have symptoms which could be ascribed to lumbarization or sacralization, although a child is occasionally seen with symptoms suggestive of hip-joint or genito-urinary disease in whom the only discoverable abnormality is lumbarization or sacralization.

Signs.—The paucity of definite objective findings is sometimes surprising in a patient with severe symptoms, another feature which often arouses suspicions of neurosis or malingering. The lumbar region of the spine may appear unduly long if lumbarization is present, while in sacralization this region is shorter than normal and the ilio-costal space is diminished. In patients with severe unilateral trouble a scoliotic deformity away from the affected side may be found. On the other hand, cases have been recorded where the development of a scoliosis due to some other condition caused impingement of an enlarged costo-transverse process on the sacrum or ilium and so produced low back pain in a patient previously free from this complaint.

In most cases localized tenderness can be elicited on pressure over the lumbosacral region. In unilateral cases this tenderness may be more marked over the anomalous process, but in other cases, for reasons already explained, the maximum tenderness may be present on the side showing no abnormality. Movements towards the affected side are limited and painful, rotation in particular being performed with difficulty. Flexion at the lumbosacral junction is diminished, and the majority are unable to touch their toes while keeping the knees straight: even when the lumbar spine is flexed as completely as possible it only becomes flat and does not show the usual backward convexity.

Spasm of the lower spinal muscles, slight tenderness over the sciatic nerve, and cutaneous hyperæsthesia along the outer side of the leg and in the foot are present in all except mild cases, while in a few chronic cases definite wasting of the leg muscles or diminution of the tendo Achillis jerk on the affected side can be distinguished.

RADIOGRAPHIC APPEARANCES

In a child the enlarged costo-transverse process bends downwards towards the sacrum, and if the condition is bilateral an appearance resembling a flattened arch is produced (*Fig.* 121). Rarely distinct evidence of the composite nature of the process is afforded by the appearance of a slight gap or foramen between the transverse and costal elements which go to form the enlarged costo-transverse process. As the person grows older the process increases in size, and in bilateral cases a ' butterfly-wing ' shadow which is characteristic is cast on antero-posterior films. Radiographs show whether the process is fused completely or incompletely with the sacrum, or whether unilateral or bilateral lumbosacral transverse articulations are present. In the latter event X rays reveal if arthritic changes have occurred (*Fig.* 122). Unless stereoscopic views are taken, it is easy to imagine that the enlarged

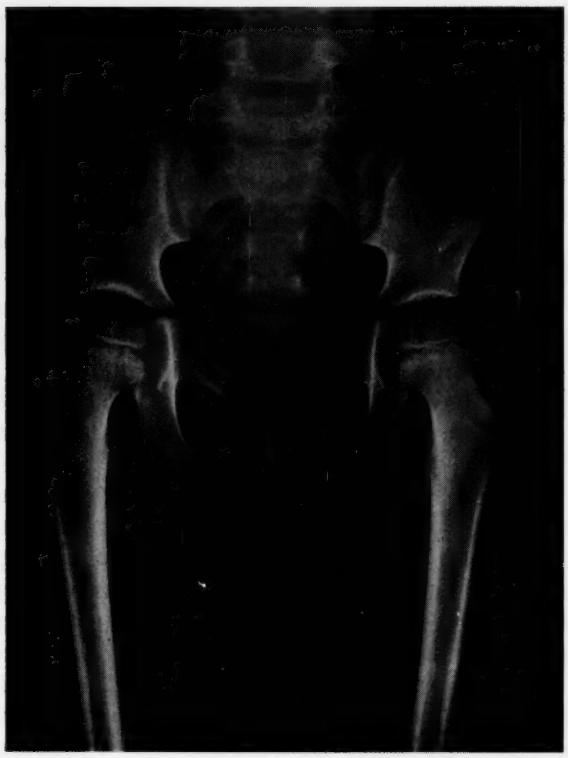


FIG. 121.—A lumbosacral transitional vertebra in a child aged eight years.

FIG. 122.—A lumbosacral transitional vertebra with bilateral costo-transverse articulations, one of which shows arthritic changes.

process is impingeing on the ilium. Actually that is unusual, although in rare instances a joint exists between the two, or the ilium may show signs of erosion through recurrent attrition by an impingeing process.

The radiographic demonstration of lumbarization or sacralization in a patient suffering from low back pain does not necessarily indicate that this developmental anomaly is the cause of the trouble. There is often a temptation to attribute the symptoms to the abnormality because diagnosis of low back conditions is never easy, and one is inclined to be satisfied if any possible cause is discovered. But the process should not be incriminated until all other causes have been considered and excluded.

TREATMENT

Conservative Measures.—These should be adopted in the first instance for all cases of lumbarization or sacralization producing symptoms, and the treatment resembles that usually advocated for lumbosacral strain.

If the low back pain is acute, rest in bed is necessary in the early stages, and any period from two to six weeks, depending on the acuteness of the case, is not considered excessive. The patient should lie on a firm bed, with soft pillows under the knees and lumbar spine. During the early stages sedatives may be necessary, and hot fomentations or cold compresses are often soothing. Massage and other physiotherapeutic measures, if employed with skill and discretion, relieve discomfort and stiffness and expedite repair. Thus ultra-violet and infra-red radiation, radiant heat, etc., have been found to give good results in the relief of pain, and at a later date ionization or diathermy may be employed with benefit. Graduated passive and active movements should be begun not later than the end of the first week, and should be continued cautiously till full movements are allowed in the sitting or recumbent position by the end of two to four weeks, the time factor depending on the acuteness of the case. These movements prevent the formation of peri-articular adhesions which are often the cause of persistent pain and disability. Incorrect posture should be corrected, and if it is due to any remediable cause this must be treated. In the severer cases the patient should be provided with a plaster jacket or a properly fitted back-brace when he is allowed up. In females strong specially-made corsets often provide sufficient support and are much more acceptable than a brace. The corsets or back-brace should be worn during the day for three months, but the prolonged use of a brace should be discouraged unless in exceptional cases.

Operative Treatment.—When conservative measures have been faithfully applied and have failed to cure the patient, operative treatment must be considered, but only a small proportion of cases require surgical intervention. Manipulation, which is often so valuable in relieving persistent or recurrent pain following lumbosacral strain, is of doubtful value in lumbarization and sacralization, as the forced movement may aggravate the condition; this is readily understandable when one considers the mechanism of pain production in these cases. Various operations have been suggested, most of which have aimed at the removal of the offending bony process, and the fact that their performance has been attended with success in many cases is strong confirmation of the correctness of the diagnosis. A few operators have found that removal of the outer part of the enlarged costo-transverse process is all that is necessary. From this they argue that the pain must have been

caused by impingement of the process on the sacrum or ilium, or by the levering effect thereby induced, and not through any pressure or tension on nerves. The offending process may be approached from behind either by splitting the erector spinæ or by working down along its outer side. This operation is not easy and hæmorrhage may be troublesome. Verrall has suggested a better method. He cuts a window in the ilium opposite the enlarged process, and through this he removes as much of the process as necessary, a method which has the advantage of removing the part of the ilium on which the enlarged process may impinge, in addition to the abnormal process itself. Other surgeons state that removal of the process tends to weaken the lumbosacral region, and argue that as nature is trying unsuccessfully to fuse the parts the aim of the surgeon should be to assist nature by performing a lumbosacral fusion operation. Although this argument is based on incomplete knowledge, the performance of the operation has given good results in many cases.

A problem arises if this type of congenital anomaly is found by chance in a radiograph. Possibly the best thing is to say nothing about it to the patient, as in many cases it never causes any trouble. Exceptions might be made to this rule in the case of people who perform extremely arduous work, or athletes who indulge in vigorous sports which markedly increase the strains and stresses borne at the lumbosacral junction. The former should be advised to find lighter work if they can possibly do so, while the latter should be counselled to avoid strenuous games.

In certain American works the spine of every new workman is X-rayed as a routine, and if lumbarization, sacralization, or other congenital defects of the lumbosacral region are found which are likely to lead to subsequent trouble, he is not put in a department requiring severe physical exertion. This type of examination, if performed regularly in Britain, would prevent a great number of the medico-legal difficulties which are prone to arise in cases of low back pain and persistent disability.

BIBLIOGRAPHY*

ABADIE, J., *Bull. et Mém. Soc. nat. de Chir.*, 1924, l, 1054.
ADAMS, Z. B., *Amer. Jour. Orthop. Surg.*, 1910–11, viii, 229.
ALESSANDRI, P., *Riv. di Clin. Med.*, 1927, xxviii, 96.
ANCEL, P. and SENCET, L., *Jour. de l'Anat. et Physiol.*, 1902, xxxviii, 217.
ANDERSON, E., *Forts. a. d. Geb. d. Röntgenstrahlen*, 1926, xxxiv, 491.
AYERS, C. E., *New Eng. Jour. Med.*, 1929, cc, 592.
BANKART, A. S. B., *Brit. Med. Jour.*, 1921, i, 587, 636.
BARDEEN, C. R., *Anat. Anzeiger*, 1904, xxv, 497.
BARDEEN, C. R., *Amer. Jour. Anat.*, 1905, iv, 265.
BASSET, A., *Bull. et Mém. Soc. nat. de Chir.*, 1925, li, 1078.
BAUMAN, G. I., *Jour. Bone and Joint Surg.*, 1924, vi, 909.
BAUMAN, G. I., *Ibid.*, 1923, v, 579.
BAUR, *Jour. of Morph.*, 1890–91, iv, 331.
BEADLE, O. A., *The Intervertebral Discs*, 1931, (*Med. Research Council Special Report Series* No. 161).
BENASSI, G., *Anthropologie*, 1927, xxxvii, 125.
BONNOIT, A., *Lyon Chir.*, 1921, xviii, 445.
BERRY, J. M., *Arch of Surg.*, 1925, xi, 883.
BEST, R. R., *Surg. Gynecol. and Obst.*, 1927, xlv, 485.
BILLINGTON, R. W., WILLIS, T. A., and O'REILLY, A., *Jour. Bone and Joint Surg.*, 1928, x, 290.
BIRMINGHAM, A., *Jour. Anat. and Physiol.*, 1891, xxv, 526.

* This bibliography is not exhaustive, but contains the majority of the more important contributions to the problem.

BOHART, W. H., *Jour. Amer. Med. Assoc.*, 1929, xcii, 698.
BOWKER, C., *Med. Jour. of Australia*, 1927, ii, 808.
BRACALONI, E., *Radiol. med.*, 1924, xi, 661.
BRACKETT, E. G., *Jour. Bone and Joint Surg.*, 1930, xii, 325.
BRACKETT, E. G., BAER, W. S., and RUGH, J. T., *Jour. Orthop. Surg.*, 1921, iii, 507.
BRAILSFORD, J. F., *Brit. Jour. Surg.*, 1929, xvi, 562.
BRASH, J. C., *Jour. Anat. and Physiol.*, 1914–15, xlix, 243.
BROWN, L. T., *Jour. Bone and Joint Surg.*, 1932, xiv, 157.
BUSTOS, F. M., *Semana méd.*, 1926, i, 144.
CAREY, E. J., *Radiology*, 1927, ix, 219.
CHANDLER, F. A., *Jour. Amer. Med. Assoc.*, 1929, xciii, 1447.
COCHRANE, W. A., *Brit. Med. Jour.*, 1928, ii, 696.
DWIGHT, T., *Anat. Anzeiger*, 1901, xix, 321, 337.
DWIGHT, T., *Ibid.*, 1906, xxviii, 33, 96.
FISCHEL, A., *Anat. Hefte*, 1906, xxxi, 459.
FRETS, G. P., *Morphol. Jahrb.*, 1911, xliii, 449.
FREY, H., *Zentralb. f. Chir.*, 1930, lvii, 2898.
GHORMLEY, R. K., *Proc. Staff. Meet. Mayo Clinic*, 1931, vi, 112.
GHORMLEY, R. K., *Collected Papers of the Mayo Clinic and Mayo Foundation*, 1930, xxii, 670.
GOLDTHWAIT, J. E., *Jour. Orthop. Surg.*, 1920, ii, 416.
HANSON, R., *Acta chir. Scand.*, 1926, lx, 309.
HEISE, H., *Deut. Zeits. f. Chir.*, 1930, ccxxvii, 349.
HENDERSON, R. B., *Brit. Med. Jour.*, 1925, i, 922.
HIBBS, R. A., and SWIFT, W. E., *Surg. Gynecol. and Obst.*, 1929, xlviii, 604.
JONES, R., and LOVETT, R. W., *Orthopædic Surgery*, 1923. London: Oxford Medical Publications.
KEITH, A., *Brit. Med. Jour.*, 1923, i, 451, 499, 545, 587, 624, 669.
KLEINBERG, S., *Med. Jour. and Record*, 1929, cxxx, 133.
LACKUM, H. L. VON, *Jour. Amer. Med. Assoc.*, 1924, lxxxii, 1109.
LE DOUBLE, A. F., *Gaz. méd. du Centre*, 1912, xvii, 29.
LÉRI, A., *Jour. méd. franc.*, 1924, xiii, 265.
LÉRI, A., *Presse méd.*, 1925, xxxiii, 1681.
LITTLEJOHN, C. W. B., *Jour. Coll. Surg. of Australasia*, 1930, iii, 201.
MACKY, K. S., *Med. Jour. of Australia*, 1927, Supp. x, 303.
MARTYN, G., *Jour. Amer. Med. Assoc.*, 1924, lxxxiii, 1297.
MENNELL, J., *Backache*, 1935. London: J. & A. Churchill.
MEYNADIER, E., *Thèse de Paris*, 1923.
MILLS, N., *Boston Med. and Surg. Jour.*, 1921, clxxxiv, 659.
MILTNER, L. J., and LOWENDORF, C. S., *Jour. Bone and Joint Surg.*, 1931, xiii, 16.
MITCHELL, G. A. G., *Ibid.* 1934, xvi, 233.
MITCHELL, G. A. G., *Lancet*, 1936, i, 75.
MOORE, B. H., *Jour. Bone and Joint Surg.*, 1923, v, 212.
MOORE, B. H., *Ibid.*, 1925, vii, 271.
MÜLLER, A., *Münch. med. Woch.*, 1928, lxxv, 695.
NISEGGI, C. H., *Semana méd.*, 1929, ii, 456.
NUTTER, J. A., *Canad. Med. Assoc. Jour.*, 1925, xv, 1055.
O'FERRALL, J. T., *Jour. Amer. Med. Assoc.*, 1928, xci, 532.
O'REILLY, A., *Jour. Orthop. Surg.*, 1921, iii, 171.
O'REILLY, A., *Jour. Amer. Med. Assoc.*, 1927, lxxxix, 1128.
OSGOOD, R. B., *Boston Med. and Surg. Jour.*, 1923, clxxxix, 1059.
OSGOOD, R. B., *Jour. Bone and Joint Surg.*, 1927, ix, 667.
PYLE, E., *New Eng. Jour. Med.*, 1931, cciv, 1083.
RICH, E. A., *Jour. Bone and Joint Surg.* 1928, x, 415.
ROHRBACH, A., *Münch. med. Woch.*, 1929, lxxvi, 592.
ROSENBERG, E., *Morphol. Jahrb.*, 1907, xxxvi, 609.
RUGH, J. T., *Jour. Bone and Joint Surg.*, 1923, v, 235.
RYERSON, E. W., *Ibid.*, 1932, xiv, 154.
SCHAUFFER, R. M., *Jour. Amer. Med. Assoc.*, 1930, xcv, 1717.
SHORE, L. R., *Jour. Anat.*, 1930, lxiv, 206.
SMITH-PETERSEN, M. N., *Jour. Bone and Joint Surg.*, 1924, vi, 819.
STRATHY, G. S., *Med. Jour. of Australia*, 1925, ii, 68.
TOYODA, J., *Folia anat. japon.*, 1927, v, 37.
VERRALL, P. J., *Brit. Med. Jour.*, 1924, ii, 798.
WARNER, F., *Internat. Jour. Surg.*, 1925, xxxviii, 179.
WILLIS, T. A., *Amer. Jour. Surg.*, 1929, vi, 163.

THREE CASES OF FRACTURE RESULTING FROM ELECTRIC SHOCK

By H. JACKSON BURROWS, London

Fractures resulting from electric shock do not appear to be generally recognized.

Cases in the Literature.—Jellinek[4] described two instances of bony trauma in men who had sustained electric shocks from lamps without suffering any mechanical violence.* The first, who experienced severe pain in the shoulder, showed radiologically a fissure 1 cm. long in the head of the humerus. The second patient, who had similar severe pain, committed suicide the day after the injury, and at autopsy the head of the humerus was found divided into numerous fragments.

Martin[6] referred to a man in whom, after contact with a current at 200 volts, it was found that the head of the right humerus "had been knocked off at the anatomical neck" and "had been displaced backwards and completely turned round". It is not stated whether mechanical violence occurred.

Frühmann[3] reported the case of a woman, aged 34, who received an electric shock from a lamp without sustaining any mechanical violence. A radiograph showed forward and downward dislocation of the shoulder with avulsion of the greater tuberosity. During a careful attempt, under ethyl chloride anæsthesia, to reduce the dislocation by Kocher's method a further fracture was produced (site not stated).

Simpson-Smith[8] described the case of a male patient, aged 41, who grasped the terminals of conductors of a 240 volt alternating current in either hand, contact being maintained for three-quarters of a minute. The head of each humerus was dislocated backwards; the left greater tuberosity was fractured, and the upper end of the right humerus was comminuted, the mushroomed head lying free.

Palugyay[7] described spontaneous fracture of the proximal phalanx of the third finger of a boy, aged 12, occurring on the fifty-fifth day after prolonged (? several minutes) contact with a wire carrying direct current at 110 volts. Other changes—Sudek's atrophy, periosteal new bone formation, and sequestration of necrotic fragments—were present in this and a similar case.

Ziemke[10] described fractures of the skull and of the middle of the humeral shaft, the bone in the latter region being found at autopsy to be so brittle that it broke like glass. The current in this instance, however, was one of 3000 volts; there was extensive charring of the soft tissues and bone, so that heat may have been a factor in the production of the abnormal brittleness. Mechanical violence also was a complication.

---

* The term 'mechanical violence' is used in the restricted sense of that of blows from falling and the like; it does not comprehend intrinsic violence such as that of uncontrolled muscular contraction.

Blencke[2] stated, without giving details, that he knew of a case in which radiographs showed distinct fissures in the distal end of the shaft of the radius after the entry of a high-tension current through the finger-tips; complaint was made of local pain.

The same author[2] and Wagner[9] describe a case of Kienböck's malacia of the semilunar bone following an electric shock from a "high-tension" current. A stone-breaker, aged 26 according to Blencke or 30 according to Wagner, suffered electric burns of three fingers from contact with the switch of a lift; he fell and lost consciousness. The right wrist became swollen and was still so two years later when it was X-rayed on account of pain experienced during hard work. The radiographs revealed the presence of Kienböck's malacia of the semilunar bone, which was ascribed to the passage of the current, though clearly it might also be ascribed to the fall at the time of the shock or be considered to have no relationship to it. The case is included here because Kienböck's disease is sometimes considered to imply a crush fracture of the semilunar bone.*

Fractures are familiar in injuries from lightning. Jex-Blake gives a review of this subject.

CASE REPORTS

Three cases, all affecting the upper end of the humerus, have been observed by the author.

Case 1.—**Crush Fracture of the Head of the Humerus.**†

A man aged 48, a hairdresser, complained of pain in and stiffness of the right shoulder. Nine and a half weeks previously, when holding a vibro-massage machine in his right hand, he was reaching with his left to pick something off the marble slab of a sink when he received a shock from a 50 cycles alternating current of 230 volts. His hands were dry; he does not think he touched a tap; the soles of his shoes were of crêpe rubber and he was standing on linoleum. He was unable to drop the machine. He felt agonizing pain all through him, and as though his right arm were being pulled out of his body. He made facial contortions but did not cry out. He felt dazed, but did not fall. A witness estimated the time which elapsed before the current was turned off at about five seconds. On the next day, or the following, he became bruised from above the right shoulder to below the right elbow. About five weeks after the injury he received massage, movements, and radiant heat three times weekly for a fortnight without improvement. He had suffered from no previous disability or injury; there was no history of any fall involving the shoulder.

On Examination.—Nine and a half weeks after the injury, the right shoulder exhibited a negligible range of movement and attempts were painful; the scapular muscles were wasted.

X-ray Examination.—A radiograph (*Fig.* 123) showed a crush fracture of the head of the humerus.

Treatment by graduated manipulations under anæsthesia, massage, movements, and radiant heat resulted in slight improvement only.

---

* The histological appearances of Kienböck's disease cannot be explained on a basis of fracture alone. Among a number examined microscopically, the author has seen unmistakable evidence of fracture in one, but it was impossible to say whether this had preceded or followed the more general changes.

† This case has been shown by a resident officer before the Neurological Section of the Royal Society of Medicine, and an account, varying in certain details from that here given, appeared in the *Proceedings*.[1]

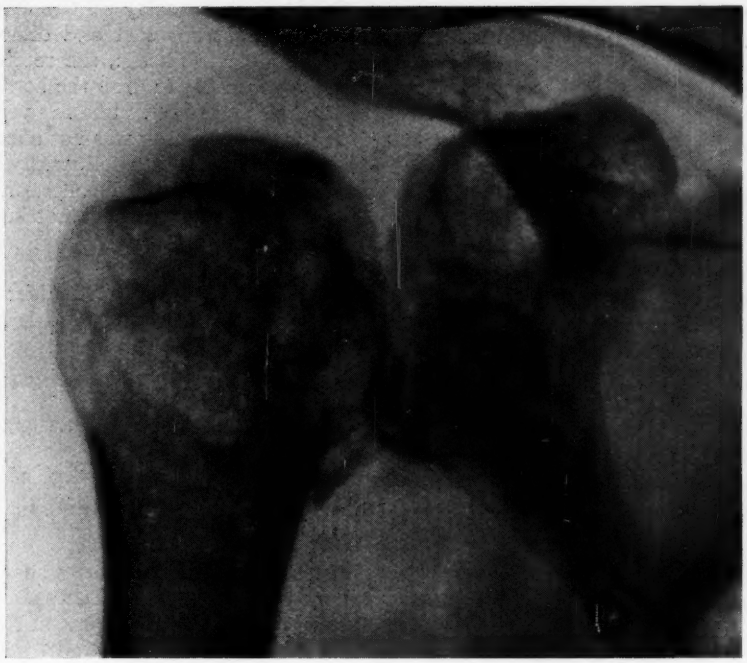


FIG. 123.—*Case* 1. Crush fracture of head of humerus.

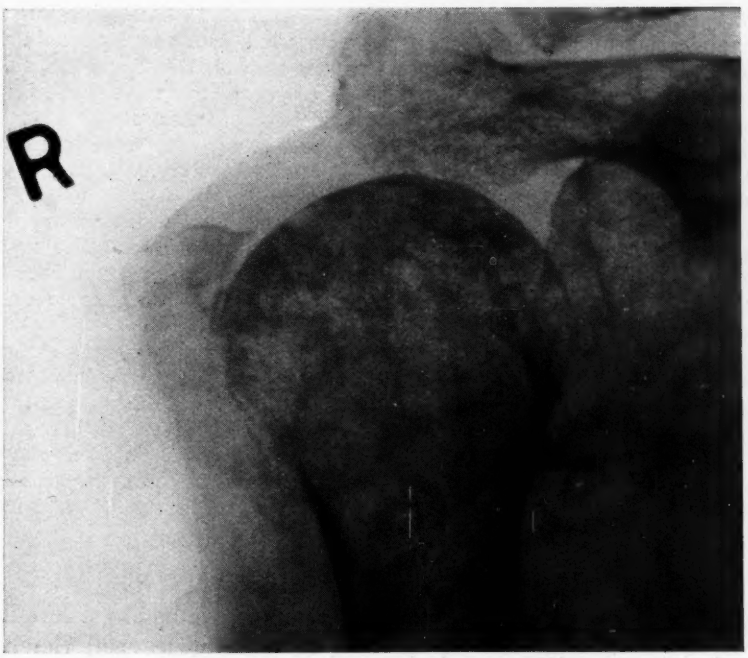


FIG. 124.—*Case* 2.—Avulsion and fracture of the greater tuberosity of the humerus.

Case 2.—**Avulsion and Fracture of the Greater Tuberosity of the Humerus.**

A housewife, aged 62, complained of pain in the right shoulder and disability of the right upper limb. The previous day, when carrying an electric kettle, she received a shock from a 50 cycles alternating current of 230 volts and was unable to drop the kettle. She felt a sudden terrible pain all over as if her arms were being dragged off. She felt very distressed and that she was about to die. She was able to scream and hearing was not affected. After an interval which she could not calculate she lost consciousness and fell, with the result, it seems, that the kettle became released or disconnected. There is no recollection of the fall. During the night the whole upper arm became much swollen. She does not know whether her hands were wet or dry at the time of the accident or what shoes she was wearing; the floor covering was linoleum.

On Examination.—Considerable limitation of movement of the right shoulder was found: the range of abduction was about 30°, scarcely any external rotation was possible, and internal rotation was limited by about 20°. There was no pronounced tenderness.

X-ray Examination.—A radiograph (*Fig.* 124) showed avulsion and fracture of the greater tuberosity of the right humerus.

Treatment.—In view of her age, treatment was confined to early massage, movements with exercises, and radiant heat.

Progress.—The fracture subsequently united, but, with the exception of a range of abduction of about 45°, no improvement in movement was found five months after the injury. The shoulder was nevertheless reasonably comfortable.

Case 3.—**Crush (Impression) Fracture of the Head of the Humerus.**

A man aged 47, a shorthand-typist, complained of pain in and stiffness of the right shoulder. Eight weeks previously, when in the loft of his house with a metal standard lamp alight in his left hand, he stumbled and touched a water tank with his right hand to steady himself. He immediately received an electric shock, and was unable to release the edge of the tank from his grasp. He experienced a 'terrible shaking sensation'; and, though he tried to shout, was unable to do so. He lost consciousness after an interval which he could not estimate. When he regained consciousness, contact had ceased, and he now experienced severe pain in the right shoulder and was unable to use the right arm; his chin and the left side of his face were cut. He was in the loft for less than five minutes altogether, but it is unknown for what fraction of this time he remained in circuit. The current was at 240 volts and alternating at 50 cycles. The tank was connected with the household water-supply. The patient's hands were dry. The right shoulder remained stiff and painful, and in the first 24 hours became much bruised. A radiograph (*Fig.* 125), taken five days later at a hospital, was considered to show no bone injury; a diagnosis of 'bruised arm,? hæmarthrosis' was made, and a course of radiant heat and massage was given, with some improvement in the pain. An examination under gas, forty-five days after the injury, was said to have revealed 'good and free' movement.

On Examination.—Eight weeks after the injury there was found to be no appreciable range of movement in the right shoulder, although attempts were painful. The scapular muscles were wasted; the circumference of the right arm was three-quarters of an inch less than that of the left, the right forearm a quarter-inch less than the left. No displacement of the humeral head was apparent.

X-ray Examination.—The old film taken five days after the injury was available (*Fig.* 125). In this projection, the overlap of humeral head and glenoid and the duplication of part of the humeral articular contour are clearly pathological; stereoscopic films were therefore taken. These appeared to show posterior dislocation of the humeral head, which was not in accordance with the clinical findings. A superior view (*Fig.* 126), however, showed the true state of affairs, namely an impression fracture of the humeral head opposite the glenoid.

Treatment.—Arthrodesis of the joint is under consideration.

DISCUSSION

Of the previous cases in the literature, there are five having a certain similarity: the two of Jellinek, those of Martin and of Frühmann, and the bilateral case of Simpson-Smith. In each instance the current appears to have been that of

FIG. 125.—*Case* 3. Antero-posterior view. Radiograph taken five days after injury.

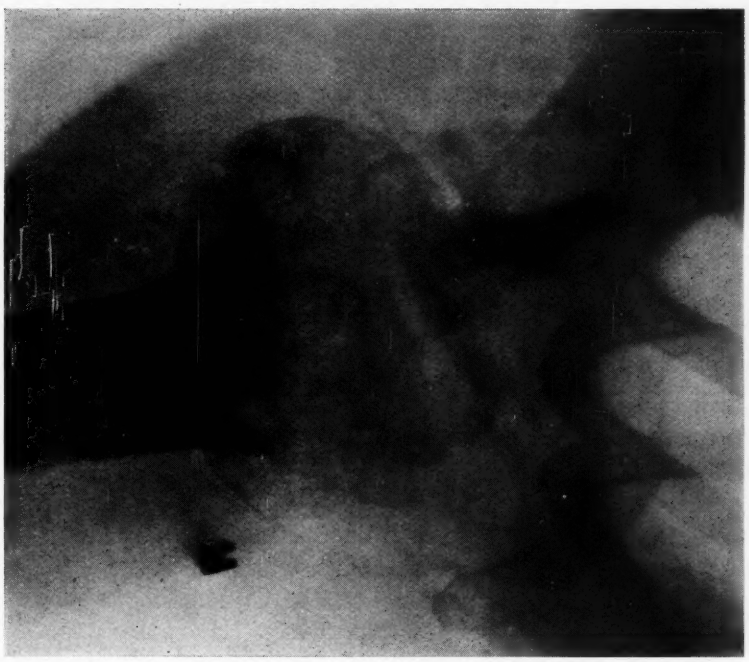


FIG. 126.—*Case* 3. Vertical view. Impression fracture of head of humerus.

a domestic supply or of a similar order of voltage; in each there has been a fracture of the upper end of the humerus, with or without dislocation. The fractures include fissuring (1 instance), comminution (2 instances), separation of the humeral head (2 instances), and separation of the greater tuberosity (2 instances). These five cases may be compared with the three described in the present paper.

It is clearly necessary in examples such as these to distinguish those in which mechanical violence can be eliminated and those in which the patient has fallen, so that the force of the fall may have been the sole or principal agent in causing the fracture. *Case* 1 is an example of the first group, in which mechanical violence can be excluded. *Cases* 2 and 3 are examples of the second, and we do not know to what extent, if any, the patient's fall may have contributed to the production of the fracture. In *Case* 3 it seems unlikely that there was any relationship, because the superficial injuries from the fall occurred on the other side of the body and because we are unfamiliar with impression fractures of the humeral head as a consequence of mechanical violence.

It is interesting to speculate on the manner in which an electric shock can cause a fracture in the absence of mechanical violence. Jellinek[3] showed that the passage of electric currents through bones, accidentally in man or experimentally in animals, may produce extremely delicate lines or planes of cleavage. These may be straight or zig-zag, and vary in length from a few millimetres to several centimetres. They can only just be distinguished with the naked eye, and are frequently not apparent in radiographs. Their presence was confirmed by histological examination, which failed to show any evidence of the effects of heat. Jellinek refers to the phenomenon as 'scission' or 'schisis'. The fact that bony injury of the more gross type inferred by the term 'fracture' is almost confined to the shoulder region and may be associated with crushing of part of the humeral head, or with dislocation, or with separation of the greater tuberosity, suggests that the principal agent is the uncontrolled contraction of the shoulder muscles. Whether direct fissuring of the bones by the passage of the current, as Jellinek suggests, may be present also and render them more susceptible to the muscular violence is a matter on which we have no information.

SUMMARY

1. Previously described cases of fracture resulting from electric shock are summarized.

2. Three additional cases which have been under the author's observation are described.

3. The mechanism of the direct production of fractures by electric shock is very briefly considered.

The author wishes to express his grateful thanks to Dr. Hinds Howell, who referred *Case* 1 to him; to Mr. S. L. Higgs, who had charge of *Case* 3; and to Dr. Russell Reynolds, Dr. J. R. Wylie, and Dr. C. M. Roberts for the use of the radiographs in *Figs.* 123, 124, and 126 respectively. He is also under an obligation to the Librarian of the Royal Society of Medicine for his assistance with the literature.

REFERENCES

[1] ALCOCK, N. S., "Electrical Injury of the Shoulder", *Proc. Roy. Soc. Med.*, 1935, xxviii, 1523 (Neurol. Sect.).

[2] BLENCKE, A., "Ein weiterer Beitrag zur Lunatumnekrose", *Arch. f. Orthop. u. Unfall-Chir.*, 1932, xxxi, 188.

[3] FRÜHMANN, P., "Die Gefahr bei Behandlung von Knochenverletzungen nach elektrischen Unfällen", *Zentralb. f. Chir.*, 1931, lviii, 1815.

[4] JELLINEK, S., "Roentgenologische Knochenaufnahmen bei Behandlung elektrischer Unfälle", *Wien. med. Woch.*, 1929, lxxix, 543.

[5] JEX-BLAKE, A. J., "Death by Electric Currents and by Lightning", Goulstonian Lectures, No. iii, *Brit. Med. Jour.*, 1913, i, 548.

[6] MARTIN, "Ueber elektrische Verbrennungen", *Zentralb. f. Chir.*, 1931, lviii, 1457.

[7] PALUGYAY, J., "Knochenveränderungen bei Verletzung durch elektrischen Strom", *Fortschr. a. d. Geb. de Röntgenstrahlen*, 1924, xxxii, 568.

[8] SIMPSON-SMITH, A., "Case of Electric Shock", *Trans. Med. Soc. Lond.*, 1931, liv, 114.

[9] WAGNER, W., "Lunatummalacie bei elektrischem Unfall", *Arch. f. klin. Chir.*, 1932, clxx, 483.

[10] ZIEMKE, E., "Todesfälle durch den elektrischen Strom und ihre Beziehungen zum Unfall", *Monats. f. Unfallheilk.*, 1923, xxx, 107.

OSTEOCHONDRITIS DEFORMANS COXÆ JUVENILIS OR PERTHES' DISEASE: THE RESULTS OF TREATMENT BY TRACTION IN RECUMBENCY *

By ARTHUR L. EYRE-BROOK

Osteochondritis deformans coxæ juvenilis, commonly known as Perthes' disease, was first described by A. T. Legg,[1] of Baltimore, in 1909. Perthes,[2] after a preliminary publication in 1910, presented a classical monograph in 1913, with the result that this disease is frequently known as Perthes' disease.

ETIOLOGY

Current views upon the etiology comprise trauma, infection, constitutional affections of bone, and abnormalities of bone development.

The Traumatic Theory has been supported by an attempt to identify the epiphysial changes following reduction of a congenital dislocation of the hip with Perthes' disease, although the former do not appear to follow the well-recognized course of the latter disease, and terminate with medial flattening, in strong contrast with the marked lateral flattening of Perthes' disease. A further point, too, is that while congenital dislocation of the hip is vastly more common in girls, Perthes' disease is more common in boys. The greater frequency in boys together with some history of recent trauma have been used in support of the traumatic theory.

Trauma may of course be only of secondary importance, as in osteomyelitis. The not infrequent bilateral disease appears to make it essential to associate the traumatic theory with some predisposition, the latter possibly being of a variable nature, as suggested by Sundt. Occlusion of part of the vascular supply to the epiphysis, the result of trauma, has been supported by a number of observers on both human and experimental pathological grounds. Zemansky and Lippmann[3] describe the results of section of the ligamentum teres in rabbits at various ages, and contend that in both animals and man the blood-vessels in this ligament normally gradually become occluded, and that trauma may result in their premature occlusion, with aseptic necrosis of the capital epiphysis. Lippmann[4] dsecribes aseptic bone necrosis in human material obtained from one of Whitman's cases.

The Infective Theory is supported by a number of distinguished observers. Platt[5] noted otherwise unexplained pyrexia in two institutional cases, although pyrexia is rarely noted in this disease. Legg[1] and Kidner[6] found staphylococci in one case each, and Phemister[7] supported the infective theory from a histological study of some human material. Against this theory are advanced the facts that no abscess formation has ever been recorded and that marked X-ray signs may be seen in many apparently clinically early cases, in marked contrast to accepted infective bone conditions.

---

* As practised at the Royal National Orthopædic Hospital (Country Branch).

The Constitutional Affections of Bone have been enlisted to explain this disease. Rickets was blamed at one time. Durham and Outland[8] note a diminution in both serum calcium and phosphorus, and support a constitutional disturbance. Sundt[9] suggests an endocrinological imbalance as the underlying factor, whilst trauma, as a rule, initiates the series of changes.

Abnormalities of Bone Development may be either of the acetabulum or of the femoral head. Murk Jansen[10] gives an ingenious explanation wherein the condition of ischium vara or of an abnormally shallow acetabulum results in point contact only between the acetabulum and the femoral head. The resultant excessive local pressure in somewhat softened bone results in Perthes' disease and deformity of the femoral head, mainly of an adaptive nature. A survey of a number of radiographs with a view to discovering any degree of ischium vara convinced me that without accurately centred X rays this could not be determined.

Delitalia[11] considered that the abnormality of bone growth was in the femoral metaphysis. He pictured Perthes' disease as similar in pathology to a slipped femoral epiphysis, save that the abnormality was transferred from the medial to the lateral region of the metaphysis.

CLINICAL PICTURE

The clinical material upon which this paper is built comprises 41 cases, varying in age of onset from 3 years and 3 months to 16 years, and showing a marked preference for the period 4–9 years, in which semi-decade lay 72 per cent of the cases. The sex incidence is male/female as 31/10; that is, the disease is three times as common in boys as in girls. Only four cases of bilateral disease were encountered.

In considering the clinical picture, those cases seen in the acute stages showed spasm, which appeared considerably in excess of the pain complained of, this always being slight. The prominence of the head in Scarpa's triangle and of the great trochanter were constantly observed, and, even after quiescence of the disease had been reached, there was usually sufficient to determine clinically the side involved. All other signs rapidly disappeared once treatment was begun.

X-ray Signs of Perthes' Disease.—These are divisible into the early signs (*Figs.* 127–130), unfortunately only too rarely seen, and the better-known typical sequence of the later stages (*Figs.* 131–135).

Early Signs.—There are five of these: (1) Increased density of the epiphysis; (2) Increased depth and clarity of the joint space; (3) Early flattening of the epiphysis; (4) Early metaphysial 'cavitation'; (5) Courtney Cages's sign.

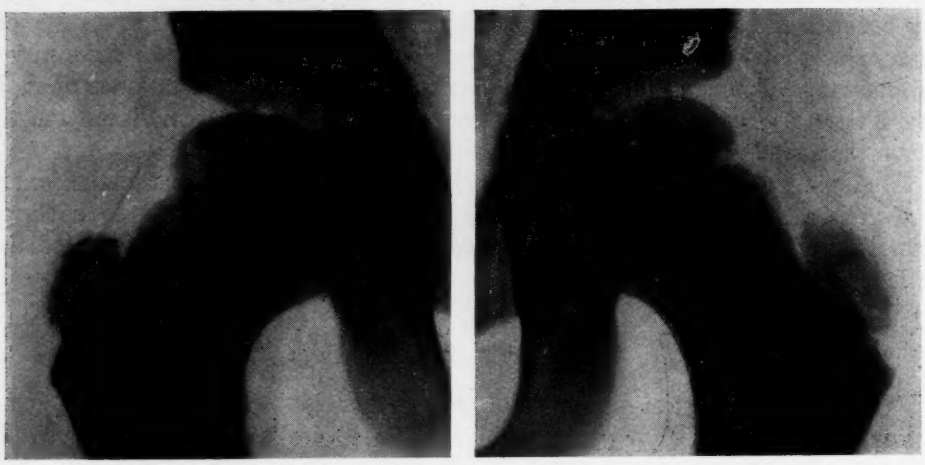
Increased density of the epiphysis appears to precede flattening, and in the earliest radiographs is only accompanied by the increase in depth and clarity of the joint space, which latter as a rule is quite a noticeable feature. These joint-space signs have been noted by a number of observers, particularly of the French school, and are presumably the result of an effusion into the joint, although the adduction of the hip does not lend support to this suggestion.

Early flattening of the epiphysis rapidly follows the above, and soon becomes one of the most striking of the X-ray signs of the disease.

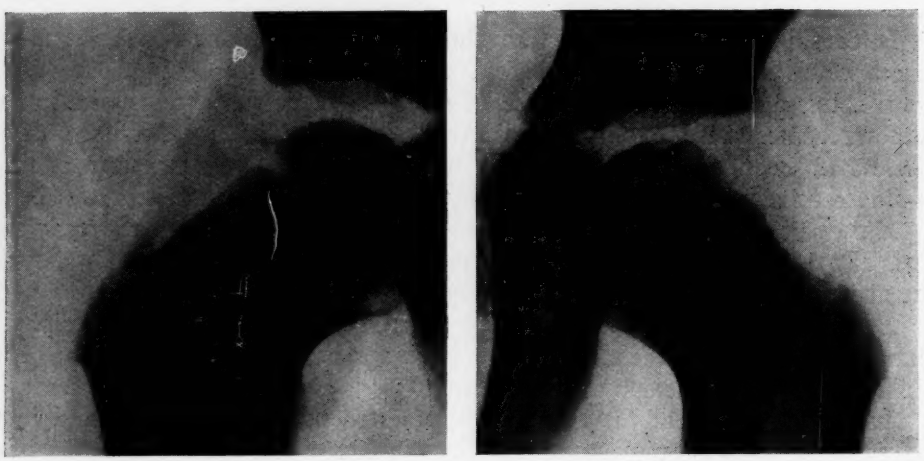
Early localized metaphysial rarefaction or 'cavitation' reminds us that this disease is not confined to the epiphysis in its manifestations. A small translucent

area in the metaphysis may be the most evident of the X-ray signs in an early radiograph, as occurred in one of our cases.

Courtney Cages's[12] sign is the lateral metaphysial erosion that develops on the affected side and contrasts with the normal straight line of junction of the metaphysis with the epiphysis. This sign was definitely present in three out of six very early cases, but does not appear to be constant or of very great assistance in early diagnosis.



FIGS. 127, 128.—*Case* 15. A. W. Aged 8 years. Perthes' disease of the right hip, showing increased depth and clarity of the joint space, increased density of the epiphysis without collapse, and Courtney Cages's sign.



FIGS. 129, 130.—*Case* 3. D. R. Aged 4 years. Perthes' disease of the right hip, showing increased density of the epiphysis without collapse, increased depth and clarity of the joint space, metaphysial 'cavitation', and a thickening of the femoral neck from subperiosteal laying-down of bone.

Later Signs.—These, the better-known signs of Perthes' disease, are (6) Flattening, fragmentation, and further condensation of the epiphysis; (7) Broadening of the neck of the femur; (8) Confluent 'cavitation' of the metaphysis; (9) Partial collapse of the 'cavited' metaphysis; (10) Regeneration of the more or less deformed epiphysis; (11) Condensation of the regenerated epiphysis;

(12) Sometimes partial disappearance of the epiphysial line in its outer portion; (13) The appearance of the transverse cervical line; (14) Adaptive acetabular changes.

FIG. 131.—*Case* 21. W. E. Aged 10 years. Much metaphysial 'cavitation'. The prognosis is bad because of the danger of severe metaphysial collapse.

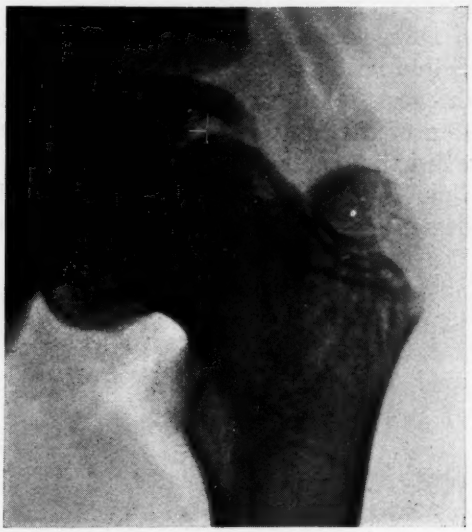


FIG. 132.—*Case* 18. A. E. Aged 7 years. Showing he formation of a marked beak, resulting in severe metaphysial deformity.

FIG. 133.—*Case* 16. B. D. Aged 9 years. Illustrating the thickening of the neck of the femur by laying-down of subperiosteal laminæ of bone along the upper surface of the neck.

FIG. 134.—W. M. Aged 9 years. Showing the transverse cervical line and the partial disappearance of the epiphysial line.

The broadening of the neck results from two processes—the laying-down of laminæ of subperiosteal bone along the superior surface of the neck, and the downward

protrusion of the inner portion of the metaphysis, this latter being known as the beak.

Severe metaphysial ' cavitation ' is a serious prognostic factor, as it is liable to result in considerable metaphysial collapse and deformity, particularly of the outer portions.

Regeneration of the epiphysis is very interesting to watch as it gradually takes visible form on the X-ray films. The outline creeps on to the upper surface of the neck, even reaching and fusing with that of the great trochanter. Any protrusion of the head beyond the acetabular margin must be taken as deformity, even when the epiphysis is agreeably round.

While the epiphysis is usually involved *in toto*, there is always a much greater degree of pathological change to the outer side. Such mushroom types as were

FIG. 135.—P. B. Aged 11 years. Like *Fig.* 134, this shows the transverse cervical line and the partial loss of the epiphysial line.

present in this series occurred as the late deformed result of former cap types which had received treatment deficient either in intensity or duration.

Partial disappearance of the epiphysial line only occurred in the more severely deformed cases, and is illustrated in one of the radiographs shown.

The transverse cervical line crosses the neck at various levels, according to the degree of deformity present, and is quite characteristic of these severer cases. It is presumably the everted edge of the metaphysis, and always starts in the beak. From the greater encroachment on internal rotation, I conclude that it is on the anterior surface of the neck.

The acetabulum shows adaptive changes after the disease has been in progress for some time. No early primary changes were noted.

TREATMENT AND PROGNOSIS

In turning to the question of treatment, we note that a great diversity of opinion has existed. Legg[13] in 1927 summed up the views of many surgeons with the remark that, " while a process suggesting weakness of bone structure is going

on, it is theoretically sound to allow no weight-bearing; but in practice, relief from weight-bearing in no way affects the end-results." I think at the present day most surgeons would at least agree with Sundt,[14] who stated that "treatment directed to the elimination of weight-bearing has no proved influence on the train of morbid changes, but its application is indicated during the stage of prominent symptoms."

The reason for the prevalence of such pessimistic views on this question is undoubtedly the advanced stage of the disease at which treatment is so often begun. The disease is so insidious that treatment is often aimed at preventing that which has already occurred. The results possible with efficient treatment of early cases, as shown in this article, make it clear that while complete freedom from all stresses "has no proved influence on the train of morbid changes", it has a very considerable influence on the results of these morbid changes.

The aim of treatment should be: (1) Clinically, to maintain a full range of movements with no predisposition to osteo-arthritis from a misfit in the acetabulum. (2) Radiologically, to produce a round head, adapted to the acetabulum, approximating as closely as possible to the normal. The radiological aim, when attained, will certainly result in clinical perfection in function of a permanent nature, but a good early clinical result may be present in a case showing considerable deformity and which is very prone to crippling arthritis.

The prognosis in this disease has been stated to depend upon: (1) The type of head—cap or mushroom; (2) The age of the patient. Most observers will not deny the influence of the following two factors also: (3) The duration of the disease before treatment is instigated; (4) The efficiency of the treatment.

I have not been able to endorse the prognostic value of the type of head as laid down by Legg[13] in 1927. That the cap type takes longer to cure than the mushroom, as he stated in 1929,[15] one must accept. It is then well on its way to consolidation, but the final result is always bad.

My series amply confirms the great importance of age in relation to prognosis, as laid down by Flemming-Møller.[16] There appear to be two factors to consider. The most important seems to be the natural course of the disease at different ages, but there is a second easily recognized factor, which is the purely mechanical one of the amount of weight carried, of the crushing forces acting on the diseased femoral head. Both these factors alter for the worse with advancing years.

The third prognostic factor is the condition of the epiphysis and metaphysis when treatment is begun. It is remarkable the degree of deformity that has often occurred by the time the child is first brought to the doctor. The head may be calcified and clearly seen, or in part decalcified and in part represented by dense calcified masses. In these latter we are still able to recognize the degree of deformity. The distance between the epiphysial line and the roof of the acetabulum gives a clear conception of the amount of collapse that has occurred in the softened head, of which the radiograph may give no outline at all. From a study of the serial radiographs of many cases, it appears to me that the flattening of these heads is permanent, and can only be relieved by some increase in depth of the epiphysis from natural growth. There does not appear to be any natural resilience or recoil to benefit by extension, nor does lateral capsular tension restore the shape.

And so we pass to the fourth point in prognosis, which is the efficiency of treatment. Treatment must be concentrated upon the avoidance of further deformity while the disease runs its course of softening, local condensation of calcium

content, fragmentation, recalcification, and consolidation. I picture each head as being in a softened condition and suffering progressive deformity from stresses, which are greater with increasing years, and I look upon the best treatment as that which will completely rid the femoral head of all stresses and allow of uninterrupted consolidation and natural growth. The treatment must relieve the head of all crushing forces, not only those of weight-bearing but also those of muscular contraction.

One other point to be considered is whether we are to encourage immobilization or movement of the affected leg. The former leads to a certain amount of wasting of muscles and stiffening of joints. The latter, while avoiding these complications, may also be of value in the treatment of the later cases by promoting a roundness of the head without any further flattening. It probably rounds the sharp edges of the head, while efficient extension preserves the maximum height in the more central portions.

With these points in mind, let us consider the methods of treatment in general use to-day. They are divisible into ambulatory and recumbent. Of the former, we have: (1) The weight-relieving calliper; (2) The patten-ended calliper; (3) Crutches, patten, and calliper.

The simple calliper merely reduces weight-bearing, but the patten-ended calliper is efficient in the abolition of all weight-bearing in the protected hip. The complete freedom from weight-bearing, however, does not protect the femoral head from all crushing forces. Trumble[17] has shown how the abductors of the hip crush the head in holding the pelvis horizontal, while moving the other leg forward. Trumble's splint therefore takes the weight centrally and dispenses with the aid of the abductors in walking, thus relieving the femoral head of all crushing forces. The third ambulatory method of treatment in common use is the calliper on the diseased side, patten on the sound side, and crutches. This fulfils all our requirements. When this system of treatment is carefully executed, the calliper and patten being worn continuously, there can be little objection to the treatment. But can this often be obtained? It will be noticed that over 72 per cent of the cases in the present series were under the age of 9 years. Can children of this age be expected to carry out this treatment in its entirety? The older children may be satisfactorily treated by this method, but in the younger children the treatment must fall very short of its theoretical possibilities.

Now let us turn to recumbent treatment and consider the methods available. These are: (1) A plaster spica; (2) Robert Jones's frame; and (3) Simple sliding traction in bed. The last of these three, and incidentally by far the simplest and cheapest, appears to meet all our requirements. The plaster spica involves much undesirable wasting and stiffening of joints, as does the Jones's frame, although to a lesser degree.

Treatment by Traction in Recumbency

At the Royal National Orthopædic Hospital the method employed consists of sliding traction with a 6 to 10 lb. weight and pulley, the foot of the bed being raised, and no splinting being used on the affected leg. Alternately Pugh's extension is perfectly satisfactory. With either method we obtain with a fair certainty our aim of reasonable freedom of movements with the avoidance of all crushing forces

acting upon the diseased femoral head. The progress is controlled by radiographs taken every 3 to 4 months, and treatment ceases when calcification and consolidation are complete. Our average duration of treatment is 18 to 24 months, but a longer period is needed in a number of cases. This treatment is such that we are confident that no known non-operative procedure can give these femoral heads a better chance of recovery. But are we obtaining this end with a minimum of expenditure and least possible interference with the normal life of the child? I believe that this is so in most cases. The children are too small to fulfil to the letter the full requirements of efficient ambulatory treatment. Education is carried out without interruption in one of our orthopædic hospitals, and the general health will certainly benefit. The need for removing the child from its home is to be regretted in most cases.

Results.—Our cases are divided into two series, those treated by extension and those otherwise treated. The former were all treated at the Royal National Orthopædic Hospital (Country Branch), where this method of treatment has been in use since 1930. In considering results, I have grouped the cases into those under 7 years and those over 7 years of age, the former to be known as *Group I* and the latter as *Group II*. Results are so dependent on the condition of the head at the inception of treatment that I have divided the original radiographs into three stages. A Stage I case presents an original radiograph showing a femoral epiphysis with little or no flattening—in fact the diagnosis may require confirmation by later radiographs. These cases can be induced to give results little short of the normal. Stage II cases show typical flattening, fragmentation, and cervical thickening. Stage III cases show more advanced changes, with recalcification.

Epiphysial Index.—For statistical purposes, I have introduced a quotient to indicate the result of treatment—

$$\frac{\text{Height of epiphysis}}{\text{Breadth of epiphysis}} \times 100 = \text{Epiphysial Index}$$

While admitting that Perthes' disease is not solely an epiphysial lesion, the metaphysial changes are nearly always proportional to the epiphysial deformity; hence the index is a fair representation of results. The normal varies somewhat with age and also from case to case. Under 7 years of age it varies from 45 to 55, while over 7 years of age it has a range from 35 to 45. It will be noted from *Table I* that with Stage I cases in *Group I*, i.e., under 7 years of age, we can approach near to the ideal of a normal head. Some slight interference with the even curve of the head is the only hint of healed Perthes' disease in some of the best results of traction treatment. The control figures do not boast of many Stage I cases in the younger group, but if we turn to Stage II cases we note an average 10 per cent improvement in the epiphysial indices in the cases treated by traction in recumbency. These control cases have been carefully treated, and many have been confined to bed for periods of six to eight months, yet there is a 10 per cent average difference in the epiphysial indices of the two series. The difference in results cannot be attributed to lack of specialization, but must be related to the treatment, the details of which are given in the table. In these younger cases the results appear amply to repay the additional trouble taken. The results are definitely superior to those obtained by other methods, and some of the very early cases give results that it would be difficult to excel.

Table I.—Results of Various Methods of Treatment in Perthes' Disease in Terms of the Epiphysial Index

	Case No.	Initials	Age at Onset	Treatment		Epiphysial Index					Treatment	Age at Onset	Initials	Case No.	
Group I, under 7 years at onset	1	L. H.	4	Extension	30 months	32	Average 39 Early Stage I	Normal 45/55	Average 26 Early Stage I	26	Plaster spica, 10/12	6	R. A.	23	
	2	J. C.	4½	,,	19 ,,	48, 36									
	3	D. R.	4	,,	14* ,,	28									
	4	O. S.	6	,,	17 ,,	48									
	5	M. B.	6	,,	18 ,,	40									
	6	D. H.	3½	,,	30 ,,	34	Average 27 Medium Stage II			30	Calliper, 3 years	3¼	J. E.	24	*Group I*, under 7 years at onset
	7	D. M.	5	,,	16 ,,	30				23	No treatment	4½	K. M.	25	
	8	T. B.	5	,,	20 ,,	30				20	Plaster and bed, 8/12; Calliper	5½	J. W.	26	
	9	A. M.	5½	,,	15 ,,	20			Average 22 Medium Stage II	25	Plaster and bed, 7/12; Calliper	5½	W. W.	27	
	10	H. H.	6	,,	17 ,,	30				25	Plaster and bed, 6/12; Calliper	6	A. D.	28	
	11	K. W.	5	,,	21 ,,	20				17	Patten-ended Calliper	5½	L. H.	29	
	12	L. C.	5	,,	12 ,,	15	Average 19 Late Stage III			16	Rest and Calliper	6	W. W.	30	
	13	P. B.	6	,,	19 ,,	29									
	14	W. M.	6	,,	6 ,,	14									
Group II, over 7 years at onset	15	A. W.	7	,,	18 ,,	36	Average 23 Early Stage I	Normal 35/45	Average 21 Early Stage I	20	Calliper, 2½ years	7	K. G.	31	*Group II*, over 7 years at onset
	16	B. D.	9	,,	18 ,,	20				22	Crutches, Patten, Calliper, 2 years	7	A. G.	32	
	17	C. C.	11	,,	13 ,,	13				21	Bed, 6/12, and Calliper	8	A. B.	33	
	18	A. E.	7			18	Average 18 Medium Stage II			25	Plaster spica, 10/12	8	R. M.	34	
	19	F. B.	7	,,	19 ,,	16			Average 18 Medium Stage II	18	Calliper, 3 years	8	L. R.	35	
	20	P. A.	9½	,,	15 ,,	16				10	Crutches, Patten, and Calliper	8	R. S.	36	
	21	W. E.	10	,,	17 ,,	15				25, 19	Crutches, Patten, and Calliper	11	V. H.	37	
	22	A. I.	11	,,	25 ,,	22				10	Calliper, 1 year	16	G. C.	38	
				,,	12 ,,	23				8, 8	Nil	9	P. B.	39	
									Average 6·6 Late Stage III	5	Calliper, 2 years	13	E. A.	40	
										5	Plaster spica, 17/12	12	B. D.	41	

* Treatment incomplete.

FIG. 136.—*Case* 4. O. S. Aged 5 years. *Group I*, Stage I case. *Treatment*: Extension for 17 months; no calliper. Radiograph showing appearance when treatment was begun. (Note early signs of the disease.)

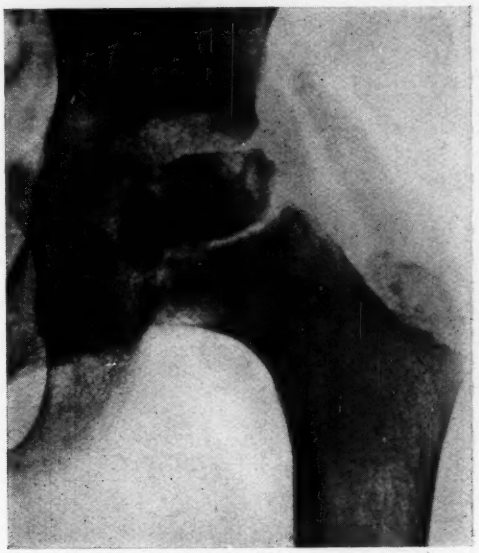


FIG. 137.—*Case* 4. After 5 months' treatment by extension.

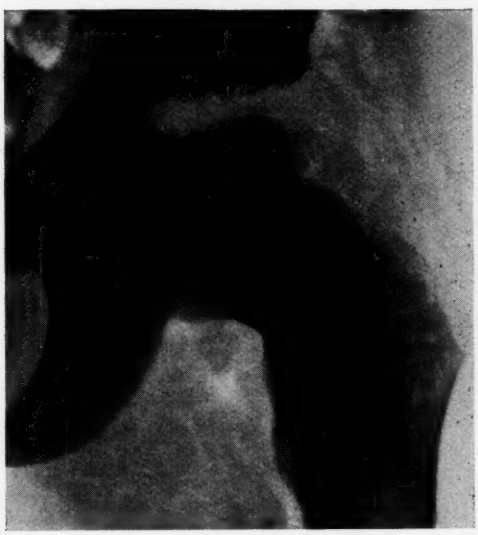


FIG. 138.—*Case* 4. After 13 months' treatment.

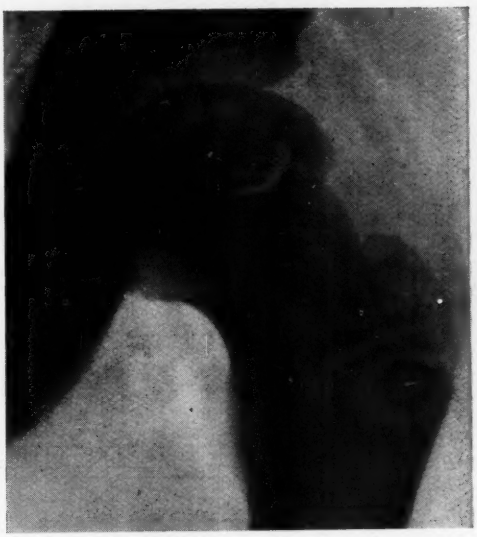


FIG. 139.—*Case* 4. Appearance 12 months after discharge. *Result*: Epiphysial Index, 48.

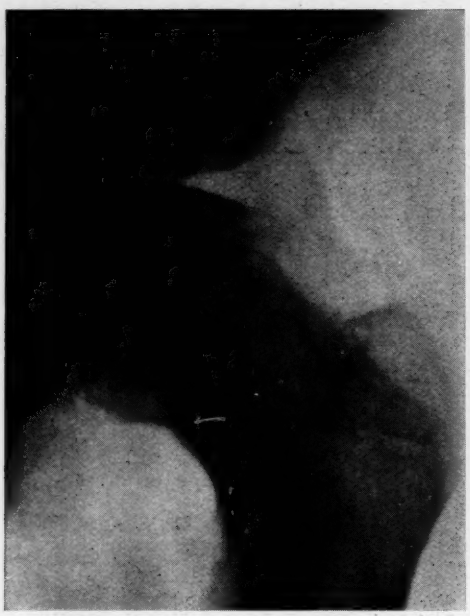


FIG. 140. *Case* 16. B. D. Aged 9 years. *Group II*, Stage I case. *Treatment*: Extension in recumbency for 18 months. Radiograph at onset of treatment

FIG. 141.—*Case* 16. After 9 months' treatment.

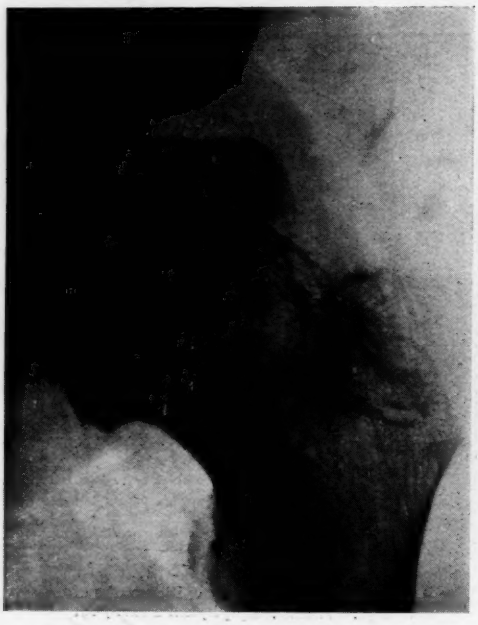


FIG. 142.—*Case* 16. After 15 months' treatment.

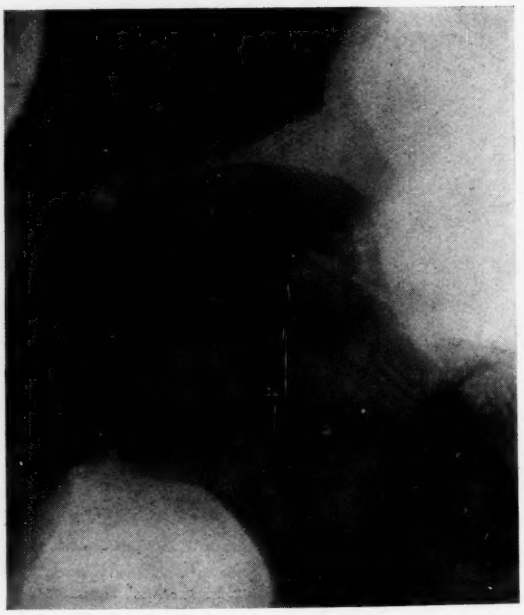


FIG. 143.—*Case* 16. After 4 years had elapsed since treatment was commenced. *Result*: Epiphysial Index, 20.

FIGS. 144, 145.—*Case* 2. J. C. Aged 4½ years. *Group I*, Stage I case of bilateral disease. *Treatment*: Extension in recumbency for 19 months. Radiographs before treatment was commenced.

FIGS. 146, 147.—*Case* 2. Radiographic appearance at conclusion of treatment. *Result*: Epiphysial Indices, R. 36, L. 48.

FIG. 148.—*Case* 5. M. B. Aged 6 years. *Group I*, Stage I case. *Treatment*: Extension for 18 months; no calliper. Radiographs before treatment was commenced.
FIG. 149.—Radiograph 12 months after treatment was completed. *Result*: Epiphysial Index, 40.

FIG. 150.—*Case* 9. A. M. Aged 5½ years. *Group I*, Stage II case. *Treatment*: Extension for 13 months and 2 months free in bed; no calliper. Radiograph at beginning of treatment.
FIG. 151.—Appearance 2½ years later. *Result*: Epiphysial Index, 20.

FIG. 152.—*Case* 10. H. H. Aged 6 years. *Group I*, Stage II case. *Treatment*: Extension for 16 months, free in bed for 1 month; no calliper. Radiograph taken when treatment was started.
FIG. 153.—Showing X-ray appearance 2½ years later. Epiphysial Index, 30.

FIG. 154.—*Case* 11. K. W. Aged 5 years. *Group I*, Stage II case. *Treatment*: Extension for 17 months, free in bed for 4 months; no calliper. Radiograph taken when treatment was commenced.
FIG. 155.—Radiograph taken 3½ years later. Epiphysial Index, 20; well-adapted head.

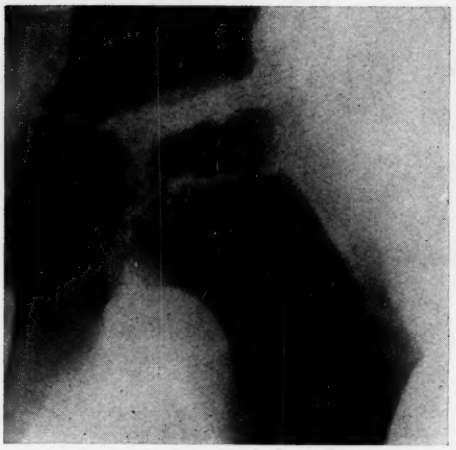
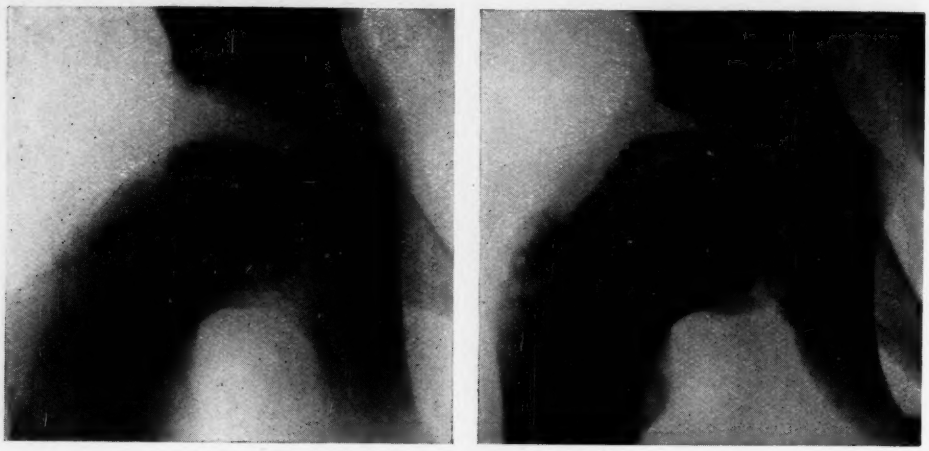


FIG. 156.—*Case* 7. D. M. Aged 5 years. *Group I*, Stage II case. *Treatment*: Extension for 15 months; no calliper. Shows X-ray appearance at start of treatment.
FIG. 157.—Radiograph taken 2 years 9 months later. Epiphysial Index, 30.

FIG. 158.—*Case* 6. D. H. Aged 3½ years. *Group I*, Stage II case (late). *Treatment*: Extension for 20 months, free in bed 10 months; no weight-bearing for a total of 30 months. X-ray photograph before treatment was started.
FIG. 159.—Radiograph at completion of treatment. Epiphysial Index, 34; head not a good shape.

FIGS. 160, 161.—*Case* 8. T. B. Aged 5 years. *Group I*, Stage II case. *Treatment*: Extension for 20 months. *Result*: Epiphysial Index, 30. Radiographs illustrate severe metaphysial 'cavitation', with resultant severe metaphysial deformity from collapse.



FIGS. 162, 163.—K. M. Aged 4½ years. *Group I*, Stage II case. *Treatment*: None; refused. *Result*: Deformed head with an Epiphysial Index of 23. The child would have had a far more deformed head had he been older, thus emphasizing the importance of the age factor. Interval between X-ray photographs, 3½ years.

FIG. 164.—P. B. Aged 10 years. This is the original radiograph of the case of a boy who had received no attention for pain in the hip and a limp, these symptoms having been present for the last 18 months. Recumbent treatment will not be repaid in any such case. Again we see the importance of age and the insidious nature of this disease.

What of the older, *Group II*, cases? Here the results are not so encouraging. In Stage I cases it appears that the results are better than those of the control series, and the index in one case actually reached within normal limits. The difference is not so marked, however, and the later cases seem to respond equally well to ambulatory methods.

From a statistical survey it appears that the results in all cases under 7 years of age and in those cases over this age in a very early stage of the disease justify the extra trouble and expense of the treatment by traction in recumbency. The

remaining cases, and in particular the late ones, certainly do not respond adequately to this treatment.

The illustrations *Figs.* 136–164 are mainly confined to some of our better results, obtained in *Group I* cases in Stages I and II of the disease. These not only illustrate the indices obtained but also the roundness of the heads, well adapted to the normal acetabulum, and thus obviating the risk of any later arthritis. Included in the illustrations are two interesting untreated cases, which demonstrate admirably the paramount importance of age in prognosis (*Figs.* 162–164). Both cases gave poor results, but the older case is far more deformed although the disease must have been in progress for a longer time in the younger case.

CONCLUSIONS

1. In considering the etiology, whether the traumatic or infective theory is favoured, it appears necessary to stipulate some predisposing factor, either constitutional affection of bone or abnormality of bone development, to account for the frequency of bilateral disease.

2. Prognosis in Perthes' disease is dependent upon: (*a*) The age of the child, which controls both the severity of the natural course of the disease and the stresses to which the diseased femoral head is to be subjected; (*b*) The stage to which the disease has progressed when treatment is begun; (*c*) The effectiveness of treatment.

3. Efficient ambulatory methods of treatment are the calliper, crutches, and patten, and the Trumble's splint. Both these are only really suitable for children over 7 years of age, and each requires constant and understanding supervision in younger children. Traction in recumbency provides the really satisfactory treatment for children under 7 years of age or over this age in a really early stage of the disease. Two methods of providing this treatment are recommended: (*a*) Sliding traction with weight and pulley; and (*b*) Pugh's extension. Results, as shown by the illustrations (*Figs.* 136–164) and *Table I*, are provided to support this contention.

4. The results are permanent, as is shown by following up some of the better results for a number of years.

5. We have never obtained a normal head, but we can approach near to this ideal.

6. Finally, I would stress the fact that the clinical results are grossly complimentary, quite severely deformed heads giving movements and function little short of the normal. We must turn to the X-ray picture and the later complications for a truer perspective. The deformed heads are all candidates for early crippling arthritis. We must not allow our 'cures' to be this fertile soil for arthritis. We must aim at permanent and anatomical and not merely temporary functional successes.

In conclusion, I wish to express my gratitude to all those surgeons in London and Bristol who gave me free access to their cases of Perthes' disease, to Mr. C. F. Coombs for helping me with the photography, and, above all, to Mr. H. J. Seddon for his help and advice in many matters.

REFERENCES

[1] LEGG, *Boston Med. and Surg. Jour.*, 1910, clxii, 202.
[2] PERTHES, *Arch. f. klin. Chir.*, 1913, ci, 779.
[3] ZEMANSKY and LIPPMANN, *Surg. Gynecol. and Obst.*, 1929, xliii, 461.
[4] LIPPMANN, *Amer. Jour. Surg.*, 1929, vi, 785.
[5] PLATT, *Brit. Jour. Surg.*, 1922, Jan., 366.
[6] KIDNER, *Amer. Jour. Orthop. Surg.*, 1916, xiv, 339.
[7] PHEMISTER, *Arch. of Surg.*, 1921, March, 221.
[8] DURHAM and OUTLAND, *Jour. Bone and Joint Surg.*, 1928, x, 301.
[9] SUNDT, *Lancet*, 1921, i, 1065.
[10] MURK JANSEN, *Jour. Bone and Joint Surg.*, 1923, xxi, 265.
[11] DELITALIA, *Amer. Jour. Orthop. Surg.*, 1915, xii, 555.
[12] COURTNEY CAGE, *Brit. Jour. Radiol*, 1933, vi, 295.
[12] LEGG, *Jour. Bone and Joint Surg.*, 1927, ix, 26.
[14] SUNDT, *Zentralb. f. Chir.*, 1920, May, 29.
[15] LEGG, *Amer. Jour. Surg.*, 1929, vi, 793.
[16] FLEMMING-MØLLER, P., *Acta Radiol.*, v, 1-36 —.
[17] TRUMBLE, *Brit. Med. Jour.*, 1935, May 25, 1070.

Literature not specifically referred to :—

LEGG, *Surg. Gynecol. and Obst.*, 1916, March, 307.
PATTISON, *Brit. Jour. Surg.*, 1928, xvi, 89.
WHITMAN, *Amer. Jour. Surg.*, 1928, iv, 185.
WHITMAN, *Ibid.*, 1929, vi, 791.
ZEMANSKY, *Ibid.*, 1928, iv, 169.
ROCHER, *Arch. franco-belges de Chir.*, 1927, xxx, 24.
LANG, *Jour. Bone and Joint Surg.*, 1932, xiv, 563.
BOZAN, *Ibid.*, 885.
SNODGRASS, *Ibid.*, 314.
CAMITZ, *Acta chir. Scand.*, 1934, lxviii, 521.
MILNER and HU, *Arch. of Surg.*, 1933, xxvii, 645.
LEWIS, *Radiology*, 1934, xxii, 188.
CALDWELL, *South. Med. Jour.*, 1934, xxvii, 402.
WALDENSTROM, *Acta orthop. Scand.*, 1934, v, 1.
BOZSAN, *Jour. Bone and Joint Surg.*, 1934, xvi, 75.
DANFORD, *Ibid.*, 516.
BRAILSFORD, *Lancet*, 1935, i, 1487.
KING, *Localized Rarefying Conditions of Bone*, 1935.

EXPERIMENTAL SURGERY

OBSERVATIONS ON THE EXPERIMENTAL PRODUCTION OF PEPTIC ULCER IN THE ILEUM

By P. P. T. WU and H. GORDON THOMPSON

DIVISION OF CLINICAL RESEARCH AND EXPERIMENTAL SURGERY, HENRY LESTER INSTITUTE, SHANGHAI

Peptic ulcer in the ileum is rare. It is practically always associated with the presence of a Meckel's diverticulum which contains heterotopic gastric mucosa in its wall.[1, 3, 6] Recent reviews of the literature by Johnson and Renner[5] and Schullinger and Stout[12] revealed the fact that there had been reported not more than fifty cases of peptic ulcer of the Meckel's diverticulum in which gastric mucosa

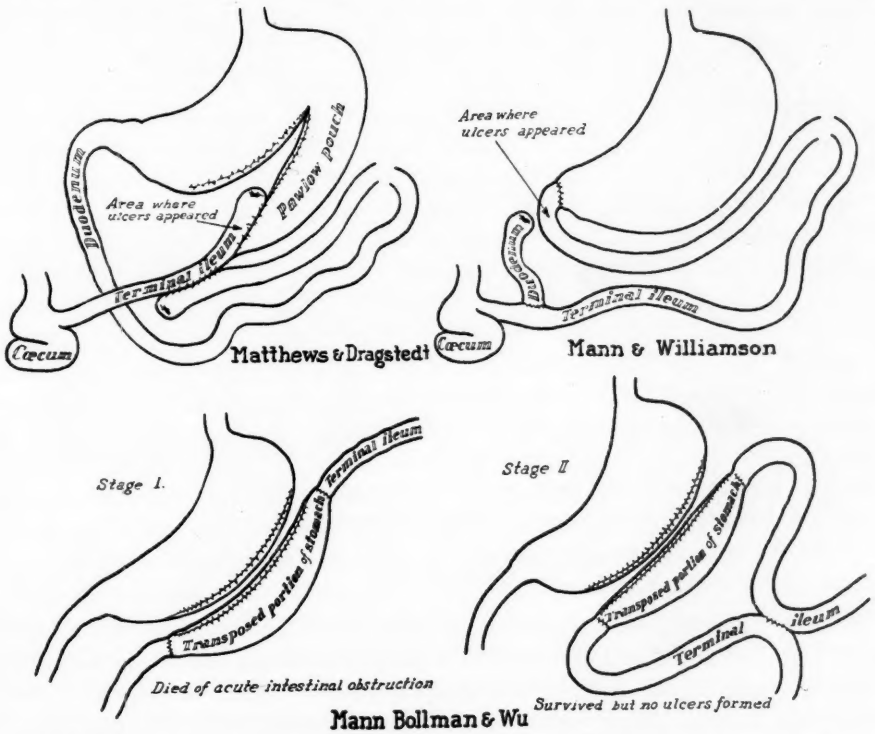


Fig. 165.—Various procedures in diagrammatic form for the experimental production of peptic ulcer.

was identified on histologic examination. As the aberrant gastric mucosa must have been present from the time of birth, it may be assumed that the development of the ulcerative process is due to the operation of certain superimposed factors. The object of this study was to reproduce the lesion experimentally and determine some of the factors concerned.

Matthews and Dragstedt[9] produced an experimental counterpart of the disease by making a small Pawlow pouch emptying into an isolated loop of the lower ileum (*Fig.* 165). In all of their six dogs large chronic ulcers were found in the intestinal mucosa adjacent to the gastric pouch between thirty-four and ninety-one days after operation.

Mann, Bollman, and Wu[7] attempted to reproduce this condition by carrying out a series of experiments on dogs in which the fundic portion of the stomach was resected and converted by partial closure into a tube. This tube formed by part of the stomach was then interpolated between the cut ends of the lower ileum by end-to-end anastomoses (*Fig.* 165). All the animals invariably died from acute obstruction, with the blockage of the intestinal contents occurring at the site of the gastro-intestinal anastomoses. Consequently, they modified the procedure by adding an entero-enterostomy between the loops of ileum immediately proximal

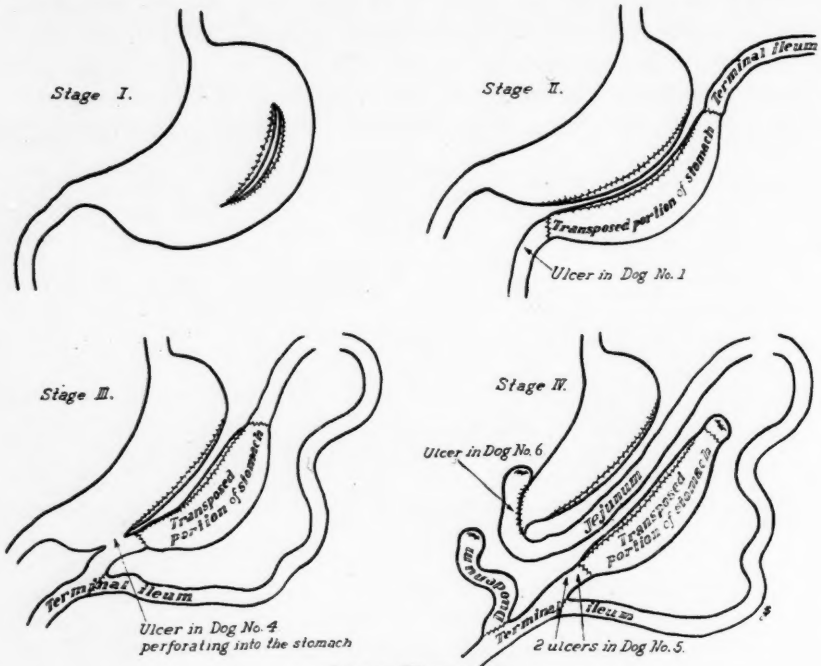


FIG. 166.—Diagrams showing the authors' procedures for the experimental production of peptic ulcer.

and distal to the transposed stomach. All the animals in this series survived, but none of them had developed an ulcer in the gastro-intestinal tract at the end of a year.

The present report is based upon the results obtained on seven of the dogs used for this study. Twenty-three operations, including three different procedures and exploratory laparotomies, were performed under inhalation ether anæsthesia and with an aseptic technique. All the anastomoses and closures of the stomach and the intestines were effected by the use of plain No. 1 catgut for the mucosa and fine silk for the seromuscular layer (*Fig.* 166).

The first two stages were really parts of a single procedure. For the first stage, the stomach along the greater curvature from the fundus to the pyloric

antrum was converted into a tube with the lumen of the latter communicating with that of the stomach proper through the two ends. After an interval of two or three weeks, the second stage was carried out, when the tube constructed from the stomach was resected and interposed iso-peristaltically between the divided ends of the ileum about 50 to 60 cm. proximal to the ileocæcal junction by end-to-end anastomoses.

It was found that by executing these procedures in stages intestinal obstruction did not occur. This is attributed to the acquisition of some sort of motor function in the tube of stomach during the interim between operations. However, fluoroscopic examination after the administration of a barium mixture failed to demonstrate any peristalsis.

Only two of the seven dogs developed an ulcer in the ileum. The first dog died from generalized peritonitis on the second day following operation. Necropsy revealed an acute perforated ulcer, measuring $3 \times 1{\cdot}5$ cm. across, on the antimesenteric side of the ileum immediately distal to the transposed stomach. The second dog, dying also from generalized peritonitis on the eighth post-operative day, showed an acute perforated ulcer, about 5 mm. in diameter, on the antimesenteric side of the ileum just proximal to the interpolated tube of stomach.

Ulcerative lesions could not be found in the remaining five dogs. One of them was killed by an accident on the fiftieth day following operation, while the other four were subjected to an exploratory laparotomy 15, 28, 35, and 55 days after the interpolation of the tube of stomach in the ileum.

In order to determine the role played by the alkaline intestinal contents in preventing the development of a peptic ulcer, a further procedure (Stage III) was carried out on each of the four dogs that survived. The ileum just above the transposed stomach was divided. The distal end was closed, while the proximal end was anastomosed to the side of the ileum about 5 to 10 cm. below the tube of stomach. It is to be noted that the experimental set-up at this time simulates in principle that of Matthews and Dragstedt.

One of the four dogs was found to have developed a chronic peptic ulcer at an exploratory cœliotomy twenty days after side-tracking the intestinal stream. The ulcer was located in the ileum just distal to the transposed stomach. It measured about 1 cm. in diameter. Its margins were indurated and its base had perforated into the wall of the stomach proper, to which it was firmly adherent (*Fig.* 167).

Exploratory laparotomies on the other three dogs 20, 28, and 34 days following the deviation of the alkaline intestinal contents from the vicinity of the transposed stomach yielded negative results.

What is the effect in the vicinity of the transposed stomach when conditions leading to peptic ulcer formation are established elsewhere? To answer this question, the classical Mann and Williamson's procedure for the production of peptic ulcer was done on the three dogs left in the series (*Fig.* 165) This consisted in replacing the duodenum by the jejunum and draining the alkaline duodenal contents into the lower ileum (Stage IV).

One of the dogs died from generalized peritonitis eight days subsequent to the last procedure. Necropsy showed two ulcers in the ileum immediately distal to the transposed stomach, but no lesion in the jejunum which was anastomosed to the stomach proper. One of them, measuring 1×4 cm. across and located on the antimesenteric side of the bowel, was perforated. The other one was

located opposite the first one and measured about 1 cm. in diameter. Another dog died on the twenty-eighth post-operative day from generalized peritonitis arising from a perforated chronic ulcer measuring about 1 cm. in diameter and located on the superior wall of the jejunum directly beyond the gastrojejunal anastomosis. The ileum on either side of the transposed stomach showed only irregular areas of superficial ulceration. An exploratory laparotomy performed on the third dog fourteen days after the establishment of surgical duodenal drainage showed no lesion in either the jejunum or the ileum.

The results of this investigation seems to suggest several things. The importance of acid as a factor in the development of peptic ulcer has been demonstrated by the experimental studies of Exalto,[2] Mann and Williamson,[8] Matthews and Dragstedt,[9] Morton,[11] McCann,[10] Wu,[13] and many others. On the other hand, the presence of heterotopic gastric tissue in the ileum only occasionally, but not invariably, leads to the development of peptic ulcer. While the difference in the outcome may depend upon a qualitative or quantitative variation of the aberrant

FIG. 167.—Photograph of specimen obtained at an exploratory laparotomy twenty days after deviating the intestinal current from the vicinity of the transposed stomach. The gastric tissue is in the middle, the proximal closed end of ileum on the right, and a chronic ulcer in the distal ileum on the left.

gastric secretion or of the ileac contents, the theory of diathesis or individual susceptibility to the disease may also be invoked. That the alkaline ileac contents exert a protective or neutralizing influence against the acid gastric juice is shown by the development of peptic ulcer following the deviation of the intestinal current from the vicinity of the transposed stomach. The fact that ulcers are not invariably produced by such procedures calls for no explanation so long as there exists the possibility of regurgitation of intestinal contents into the side-tracked portion. Finally the development of ulceration in the ileum after the operation of surgical duodenal drainage to produce a jejunal ulcer had been added to the other two procedures is somewhat suggestive of a reflex nervous or hormonal factor. Until secretory nerves to the islands of gastric tissue in a Meckel's diverticulum have been demonstrated, a hormonal mechanism like that established by Ivy and Farrell[4] in autogenous pouches of the fundic portion of the stomach transplanted subcutaneously in dogs appears to be more important from the clinical point of view.

In conclusion, it may be stated that peptic ulcer in the ileum has been produced experimentally by one or a combination of several procedures. The observations made in this study seem to lend support to the hypothesis that the development of peptic ulcer in a Meckel's diverticulum containing heterotopic gastric mucosa depends on biologic, chemical, and possibly reflex nervous or hormonal factors.

REFERENCES

[1] ASCHNER, P. W., and KARELITZ, S., *Ann. of Surg.*, 1930, xci, 573.
[2] EXALTO, J., *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, 1911, xxiii, 13.
[3] GREENWALD, H. M., and STEINER, M., *Amer. Jour. Dis. Child.*, 1931, xlii, 1176.
[4] IVY, A. C., and FARRELL, J. I., *Amer. Jour. Physiol.*, 1925, lxxiv, 639.
[5] JOHNSON, L. B., and RENNER, G., jun., *Surg. Gynecol. and Obst.*, 1934, lix, 198.
[6] LINDAU, A., and WULFF, H., *Ibid.*, 1931, liii, 621.
[7] MANN, F. C., BOLLMAN, J. C., and WU, P. P. T. Unpublished data.
[8] MANN, F. C., and WILLIAMSON, C. S., *Ann. of Surg.*, 1923, lxxvii, 409.
[9] MATTHEWS, W. B., and DRAGSTEDT, L. R., *Surg. Gynecol. and Obst.*, 1932, lv, 265.
[10] MCCANN, J. C., *Arch. of Surg.*, 1929, xix, 600.
[11] MORTON, C. B., *Ann. of Surg.*, 1928, lxxxvii, 401.
[12] SCHULLINGER, R. N., and STOUT, A. P., *Arch. of Surg.*, 1934, xxviii, 440.
[13] WU, P. P. T., *Ibid.*, 1935, xxx, 557.

SHORT NOTES OF RARE OR OBSCURE CASES

LEIOMYOMA OF THE MEDIAN NERVE

By CECIL P. G. WAKELEY
SENIOR SURGEON, KING'S COLLEGE HOSPITAL

TUMOURS of nerves are rare and always interesting to the surgeon inasmuch as they vary considerably in their symptomatology and often cause difficulty in diagnosis. The case described here was not diagnosed as a nerve tumour at all, and was thought to be a sarcoma of muscle. The following is the history of the case :—

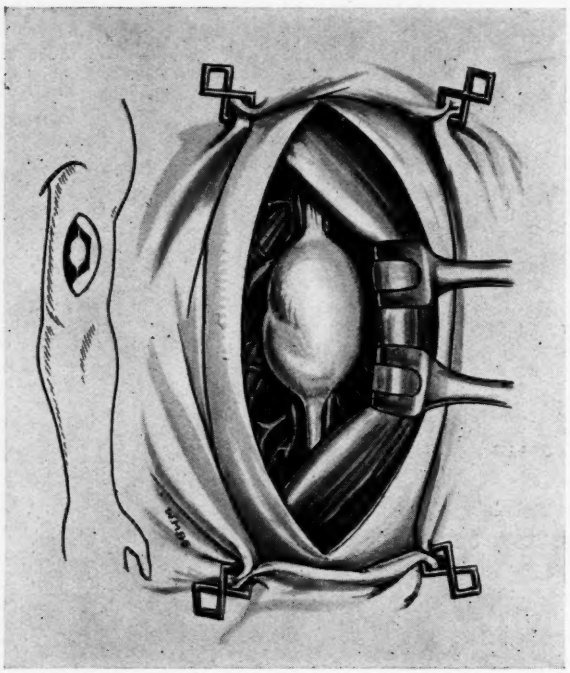


FIG. 168.—Drawing made at the time of the operation, showing the tumour situated on the median nerve. The inset shows the position of the incision in the arm.

Miss Emily D., aged 44, came under observation in October, 1935, complaining of a hard lump in the inner side of her left upper arm. She discovered the lump by accident about a year previously and it has slowly increased in size.

The swelling was hard but painless, and on palpation no painful sensations were produced in the forearm or hand. The size of the swelling was about that

of a tangerine orange; it was fixed to the deep structures of the arm, but not to the skin, which could be freely moved over it. Although fixed to the deep structures it was not fixed to the humerus, and it did not limit any movements of the arm. Sensation in the whole upper extremity appeared to be normal.

Operation was decided upon, and on Nov. 21 a longitudinal incision was made over the tumour; on incising the deep fascia it was found that the tumour was situated on the median nerve (*Fig.* 168). With some difficulty the tumour was completely excised from the nerve and all the small bleeding vessels were ligatured. The wound was closed with interrupted sutures.

FIG. 169.—The tumour bisected, showing the area of necrosis in the centre.

An uninterrupted recovery ensued, with the exception of a slight paresis of the median nerve which has since completely cleared up. The excised tumour was quite smooth on the surface but had a very hard consistency; on bisecting it a large area of central hæmorrhagic necrosis could be seen (*Fig.* 169). On histological examination the

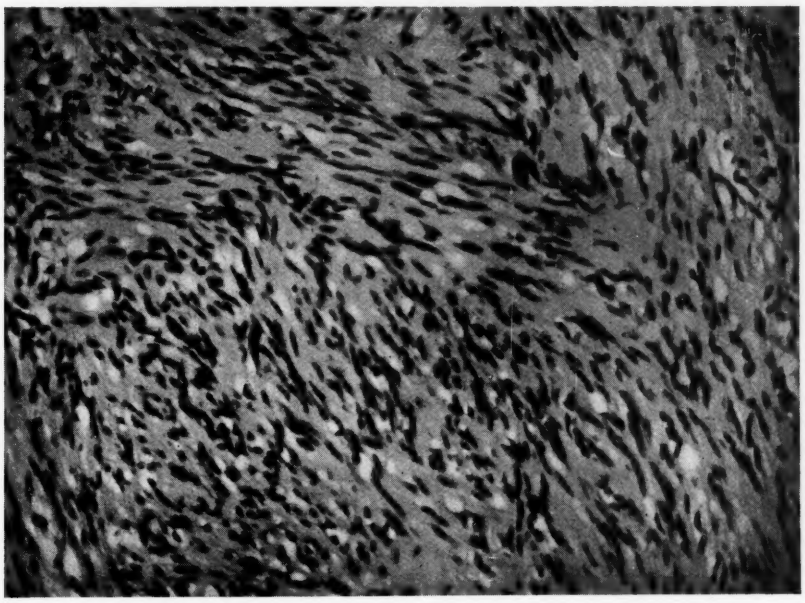


FIG. 170.—Microphotograph showing typical arrangement of the leiomyoma.

tumour was found to be a leiomyoma. It consisted of bundles of parallel smooth muscle cells. The bundles showed some tendency to a whorled arrangement (*Fig.* 170). There were areas of œdema and of hyaline degeneration.

HÆMANGIOMA OF VOLUNTARY MUSCLE

By B. W. FRANKLIN BISHOP, Kimberley

In the October, 1935, issue of the Journal there are two articles on the above condition. A further case is here recorded :—

History.—A Jewish youth, aged 19, came to see me complaining of a swelling in the lower part of his right leg. He had no idea how long it had been there, though his notice was drawn to it some two years previously. He could give no definite history of injury to that part of his leg. There was not much pain, though

Fig. 171.—Microscopical section of hæmangioma of voluntary muscle.

he stated that his leg became tired easily and that he limped slightly. He could not say whether he had had this swelling all his life or not. He apparently did not attach much importance to it.

On Examination.—There was an ovoid swelling about the size of a smallish orange situated in the lower third of his leg, posteriorly. It stopped short about

½ in. above his ankle. It seemed to be situated deeply in the muscles but was freely movable over the bone from side to side. It varied in consistency, parts appearing cystic and other parts hard. There was no pulsation either visible or on palpation. The posterior tibial artery was pulsating well. There was very definite wasting of the calf muscles, 1 in. difference as compared to the other leg. X rays revealed pressure indentation and erosion of the fibula, with minute areas of calcification in the soft tissues.

OPERATION.—A vascular dark-brown tumour was found implicating all muscles in the vicinity of the tumour. There was no capsule, and the tumour was so widespread that it was impossible to remove it *in toto*. An attempt was made to remove the margin. Portions were removed for section.

PATHOLOGICAL REPORT.—Spreading hæmangioma with islets of chronic inflammation and heterotopic bone.

Sections of the growth show the structure of a hæmangioma of the spreading type (*Fig.* 171). Foci of chronic inflammation and small islets of heterotopic bone are present in parts of the stroma.

Commentary.—In view of the rarity of these cases it seems of interest to report this example. It was impossible to state definitely from which muscle the tumour had arisen, though probably it was the peroneus longus.

RETROGRADE INTUSSUSCEPTION OCCURRING DURING LIFE

BY ALEX. MITCHELL, ABERDEEN

IN view of a report on a case of retrograde intussusception occurring during life by Mr. Emlyn Lewis in the JOURNAL of January, 1936, I think the following case might be of interest.

A girl, aged 15, was seen on Feb. 12, 1935, with a history of intermittent abdominal pain for two days. During the last few hours the pain had become very severe. No definite physical signs were made out in the abdomen, except a little fixation and tenderness on the right of the umbilicus. Pulse and temperature were normal and the patient did not look ill. The bowels had acted two days previously after castor oil.

OPERATION.—Under a general anæsthetic the abdomen was opened by a right paracentral incision. Intussusception of the ileum about one foot from the ileocæcal junction was found. The intussusception was of a retrograde type and the enclosed portion was completely gangrenous. The portion of bowel affected was about one foot long. There was no appearance of tumour or any other abnormal condition to be made out. As the intussusception was irreducible, it was resected, with closure of both ends of the bowel and a lateral anastomosis.

The patient made an uneventful recovery and left the nursing home three weeks after operation.

The condition is undoubtedly a rare one, but this case alone is sufficient to show that it is not always a post-mortem condition.

ACUTE PSOAS ABSCESS CAUSED BY DIVERTICULITIS

By ALEXANDER LYALL,
EXTRA DISPENSARY SURGEON, ROYAL INFIRMARY, GLASGOW

THE complications of diverticulitis are many, and indeed it is one of the conditions which must be constantly kept in mind in the diagnosis of obscure intra-abdominal conditions. The following case illustrates yet another of its interesting complications, and one which, as far as we are aware, has not formerly been described.

John D., aged 64 years, a steelworks foreman, was admitted to the Glasgow Royal Infirmary on Jan. 17, 1935, with the following history. Seven weeks before admission the patient developed pain in the right leg. It was diagnosed at the time by his doctor as muscular rheumatism and he received massage treatment, which, however, made the condition much worse. The thigh became more swollen and very painful and he began to have sweating and feverish attacks. Upon admission to hospital, the right thigh was markedly swollen from the inguinal region down to the knee, where the swelling gradually faded away. The skin on the outer side of the thigh showed marked pitting on pressure. Under general anæsthesia incisions were made into the outer and the inner aspects of the upper thigh, and much foul-smelling pus was evacuated. Culture of the pus showed the presence of hæmolytic streptococci, and antiscarlatinal serum was given. The patient's condition gradually deteriorated, however, and he died six days after admission.

A post-mortem was performed ten hours after death. The body was that of a well-nourished elderly-looking man. Recent operation incisions were present on the outer and inner aspects of the right thigh, the external wound containing packing and exuding a great deal of pus. The sigmoid loop of bowel showed a well-marked diverticulosis and was adherent to the brim of the lesser pelvis on the right side. When these adhesions were broken down, a necrotic diverticulum was found to be attached to the psoas sheath and to communicate with an abscess in it. The pus extended upwards along the muscle to its lumbar attachment and downwards beneath the inguinal ligament into the thigh. Here the pus was found to have ruptured through the sheath and spread down the postero-internal part of the thigh to near the knee-joint. The pus surrounded the upper part of the femur and formed a large abscess in this region which had been opened by the operation incisions. There was no disease of the femur, ilium, vertebral column, hip, or sacro-iliac joints.

The condition was obviously an acute psoas abscess which had ruptured into the thigh and was caused by an inflamed diverticulum of the sigmoid becoming adherent to the sheath of the muscle.

My thanks are due to Mr. Macewen and Professor Shaw Dunn for permission to use the clinical and pathological summaries of this case.

REVIEWS AND NOTICES OF BOOKS

An Index of Differential Diagnosis of Main Symptoms. Edited by HERBERT FRENCH, C.V.O., C.B.E., M.A.(Oxon.), F.R.C.P.(Lond.), Consulting Physician to Guy's Hospital, etc. Super royal 8vo. Pp. 1145 + xii, with 742 illustrations (196 in colour). 1936. Bristol: John Wright & Sons Ltd. 63s. net.

IT is eight years since the last edition of this work appeared, and the fact that since its first appearance in 1912 over fifty thousand copies have been distributed shows that it has been of great value. It really consists of a full descriptive text-book of medicine, surgery, and gynæcology, but all the information is arranged under symptoms and not under diseases. Its usefulness depends largely on the accuracy and completeness of the index, which occupies no fewer than 220 pages. Under the heading of pain in the back—for example—there are over ninety references in the index, and all other subjects are dealt with on the same scale. Over fifty pages are given to the diagnosis of 'swellings' in different parts of the body. The numerous illustrations are well chosen and well reproduced. Advances in recent methods of diagnosis of the biliary and urinary tracts are described, as well as the rarer diseases tularæmia, botulism, psittacosis, and pink disease. Dr. French, who has written the largest part of the book, is assisted by eighteen other contributors, of whom Mr. Ogilvie and Professor Bruce Perry take the place of the late Mr. Rowlands and Dr. Carey Coombs. It certainly forms an invaluable book of reference.

Handbook of Surgery. By ERIC C. MEKIE, M.B., Ch.B., F.R.C.S.E., Medical Officer, Malayan Medical Service. With a Foreword by JOHN FRASER, M.C., M.D., Ch.M., F.R.C.S.E., Regius Professor of Clinical Surgery, University of Edinburgh. Crown 8vo. Pp. 699 + xii, with 24 illustrations. 1936. Edinburgh: E. & S. Livingstone. 12s. 6d. net.

MR. MEKIE states the purpose of his book in the preface: "In such a handbook as this—expressly designed for the last-minute revision—little that is original can be incorporated except in arrangement, and all that is non-essential to the task of surmounting the final hurdle of the medical undergraduate career is excluded. The special subjects have therefore been omitted or rigidly abbreviated."

"In presenting this handbook I would urge the student to realize its limitations. It is but a supplement to his larger text-books, to which he must look for guidance on details and by which he should conduct his earlier studies."

The volume contains an enormous number of facts, which are clearly catalogued and in many instances are actually set out in tabular form. As one reads the book one is bewildered by the knowledge crammed into so small a space. For students revising for their qualifying examinations and also for the higher examinations in surgery, the book is excellent and should fill a most important place. One hopes—with the author—that it will be consulted only after the standard text-books have been read, and that it will not be used as the only means of acquiring a theoretical knowledge of surgery.

Elementary Surgical Handicraft. By J. RENFREW WHITE, Ch.M., F.R.C.S., F.A.C.S., Surgeon to the Dunedin Hospital, New Zealand, etc. Demy 8vo. Pp. 250 + vi, with 243 illustrations. 1936. London: J. & A. Churchill Ltd. 8s. 6d. net.

THIS small volume is produced in the hope that it may assist students and nurses in the handicraft of elementary procedures. The various methods of employing sutures, of tying knots, and of applying adhesive plaster strapping, are described with good illustrations. The pages devoted to the arrest of regional hæmorrhage will prove useful to house surgeons. The

book fulfils its mission so far as it clarifies many points which are often regarded as mysterious by the novice. In most respects we agree with the methods of approach to the uninitiated which the author adopts. The older methods of operating upon fractures and some detailed references to bone-grafting might with advantage be replaced by a description of local anæsthesia and the injection treatment of varicose veins and piles, and other minor procedures with which the student may be faced on receiving his final degrees. A revision of such a nature would be in keeping with the objects at which the author aims. It is perhaps unwise to emphasize in the preface of a book for students that surgery means handicraft. A good operator may be a menace to surgery unless he is also a first-class clinician, with instinctive judgement and alive to the necessity for strict personal attention to modern pre-operative and post-operative methods. The text, however, is confined to handicraft and contains many practical hints and suggestions.

A Synopsis of Surgical Anatomy. By ALEXANDER LEE MCGREGOR, M.Ch. (Edin.), F.R.C.S. (Eng.), Lecturer on Surgical Anatomy, University of Witwatersrand, etc. With a Foreword by Sir HAROLD J. STILES, K.B.E., F.R.C.S.E. Third edition. Crown 8vo. Pp. 664+xviii, with 648 illustrations. 1936. Bristol: John Wright & Sons Ltd. 17s. 6d. net.

THE good reception given to this book has fully borne out what we expected after reading the first edition for review. It presents a much closer association of anatomy with practical surgery than any other book we know. The section, for example, on the sympathetic, with the diagrams explaining the various operations, is a model of clearness. The present edition has a detailed description of the nerves related to the thyroid gland, of renal-adrenal adherence, and diaphragmatic hernias. There have also been numerous additions to the diagrams which make so much for the elucidation of the text.

Traité de Chirurgie d'Urgence (Félix Lejars). By PIERRE BROCQ, Professor agrégé à la Faculté de Médecine de Paris; Chirurgien des Hôpitaux; and ROBERT CHABRUT, Ancien Chef de Clinique à la Faculté de Médecine de Paris. Ninth edition. Large 8vo. One volume of 1286 pages, with 1250 illustrations. Paper covers, Fr. 170; bound, Fr. 190. In two volumes (Pp. 1299 + xv), Fr. 200. 1936. Paris: Masson et Cie.

LEJARS' *Chirurgie D'Urgence* first appeared in 1899; eight editions have already been published in France; it has been translated into many languages, and many thousands of copies have been sold throughout the world. A book with such a record has become an institution, no longer subject to the ordinary critical review; a new edition was anticipated with pleasure and welcomed on its appearance.

Chirurgie D'Urgence was Félix Lejars' master work. It was the expression of his surgical life. He said that he had lived it before he wrote it, had produced it slowly and painfully, dominated by a burning desire to lighten the burden of suffering for the sick and the injured and at the same time to help the practitioner of medicine, particularly the isolated practitioner, the hospital resident, and the young surgeon. It was with these ideals and in this spirit that the eight editions were written. Amongst the last desires of a strenuous life, Lejars had contemplated the preparation of a ninth edition. Weariness primarily, sickness subsequently, prevented the realization. It was necessary to entrust the work to other hands. The new edition appears with the names of Professor Pierre Brocq and Dr. Robert Chabrut on the title page.

When one has known the book since its first appearance and known its author, and has followed it carefully and appreciatively through the later editions and impressions, some feeling of anxiety is unavoidable when greeting the new volumes, lest it should be found that past high standards and traditions have not been adequately maintained. Let it be said at once, close and critical examination shows that there is no ground for any fears.

The present authors claim that though the book has been entirely recast and brought into touch with the very latest surgical thought and practice, they have closely followed the original plan and have striven earnestly to maintain the ideals and spirit which characterized the previous editions.

Notable changes have come about in recent years in the conditions under which medical and surgical work is carried on. Particularly is this the case with regard to means of transport

and the provision of new, adequately equipped, and well-staffed hospital centres, even in remote and sparsely populated districts. It now but rarely happens that a practitioner is left, dependent on his own resources, isolated except perhaps for the assistance of an anæsthetist, face to face with a serious surgical emergency. But though he is absolved from the necessity of actually performing the operation or carrying out the necessary treatment, though it may be easily possible to obtain skilled advice and help, though it may be a simple matter to dispatch the patient to the nearest surgical centre, it is even more important than before that he should be able to recognize early signs and symptoms and indications and be fully alive to the possibilities and limitations of surgery. Satisfactory results in urgent conditions depend largely on early intervention, which again depends on early diagnosis and action on the part of the practitioner who first sees the patient.

Following the lines laid down originally by Lejars, urgent surgical conditions are envisaged in the very widest possible sense. The book is no mere manual of minor surgery; though the needs of the general practitioner and the hospital resident are fully considered and amply provided for, it is perhaps to the young, well-trained, but relatively inexperienced general surgeon that it will make the greatest appeal and be of the greatest use. Incidentally it may be added that even the experienced surgeon will find much of value and interest. The chapters dealing with the treatment of fractures of the base of the skull, with intrathoracic wounds, injuries, and suppurations, with thoraco-abdominal lesions, and with subphrenic and hepatic abscesses are well worthy of careful consideration, together with much else.

The subject matter has been so very well brought up to date that here and there are to be found suggestions and recommendations so novel that they can hardly be accepted forthwith. Attention may particularly be directed to the chapter on the Prevention of Tetanus, which is based upon and embodies textually the Report of a Commission appointed by the Society of Surgeons of Paris to study the question. The report, dated July, 1935, received, and has since been published with, the approval of the Society. Shortly, it condemns the use of antitetanic serum except for treatment, and strongly advocates active immunization by means of antitetanic vaccination, particularly of conscripts called up for military service and of workers in certain occupations carrying risks of repeated minor injuries.

It is needless to particularize further. The same high standard is maintained throughout; the ardent spirit of Félix Lejars still pervades the pages. One may not always agree, but one must always think. *Chirurgie D'Urgence* has been and remains a great book. It is unique. To any medical man or woman, practitioner or specialist, physician or surgeon, who can read French it is to be recommended; even those who have no great knowledge of the language may follow the text in the wealth of illustrations, well-planned, well-executed, and informative. An excellent index of illustrations is a valuable, and to the reviewer a novel, feature of the work.

Post-graduate Surgery. Edited by RODNEY MAINGOT, F.R.C.S., Senior Surgeon to the Royal Waterloo Hospital, etc. In three volumes. Royal 8vo. Vol. II. Pp. 1747–3572, with 1134 illustrations. 1936. London: Medical Publications Ltd. £9 9s. the set, or £3 10s. per volume.

THE expectations aroused by the first volume of this great work have been more than fulfilled by the second. First, as to the actual contents of this volume, the mere enumeration of which is a matter of some length.

The section on the Head, Spinal Column, and Salivary Glands by Cecil Wakeley is a remarkable combination of simple, practical instruction with scientific exposition of the subject. (But why speak of 'Spinal Column' when the subject is that of the Spinal Cord?) The routine examination in cases of head injury is very clearly put. The illustrations of operations on the Gasserian ganglion and its root are beautifully clear. The whole question of tumours of the brain is made simple by the very concise descriptions and figures.

The chapters on the Neck are by Sir William Wheeler, and include a very well illustrated account of the excision of thyroglossal sinuses, and also of Henry's operation for the ligature of the first part of the subclavian artery.

McNeill Love writes on the Surgery of the Breast. He advocates and illustrates a good method of 'closed' drainage of a mammary abscess. In the description of the radical breast amputation we think that the advantages of the use of the diathermy knife might have been mentioned.

The chapters on the Thorax are an example of one of the best features of this work, because they embrace the medical as well as the surgical aspects of the subject. They are written by Sleigh Johnson and Holmes Sellors, and include post-operative chest complications, artificial pneumothorax, empyema, the surgical treatment of pulmonary tuberculosis, pulmonary embolism, and bronchiectasis. We should have thought that a work of this size might have included some account of the surgery of the heart, but perhaps this is to come in the third volume.

The section on Gynæcology is by Lyle Cameron (regional gynæcology and gynæcological operations), Sidney Forsdike (sterility), and V. B. Green-Armytage (ovary). It represents a very complete treatise on gynæcology, including an account of the investigation of the causes of sterility by radiology and other methods, and the various operations done for the relief of certain types of this condition.

Ainsworth-Davis writes on the Urinary System and the diseases of the male genital organs. This section begins with eighty-three pages devoted to the various physical and biochemical methods of examination, including the routine examinations—cystoscopy, pyelography, etc.—and the less common methods of urethroscopy and vesiculography. In relation to the diseases of the kidneys, a long account is given of the ketogenic diet, but we are told that the use of mandelic acid has now superseded this. A full account is given of von Lichtenberg's operation for the plastic repair of hydronephrosis. In the section on the prostate full accounts are given of the Harris operation for repair of the prostatic bed and of the perurethral operation. In the treatment of retained testis Ombrédanne's method of orchidopexy, in which the testis is merely anchored in the scrotum, is the only one given, and we think that the more efficient method of anchoring the testis and scrotum to the thigh (Torek) should have been mentioned. In the operation for excision of urethral stricture, no mention is made of the longitudinal splitting of the tube, a point the importance of which was demonstrated by Hamilton Russell.

A special part is devoted to the Sympathetic Nervous System, its anatomy, physiology, and clinical aspects. This is by Lawrence Abel, and is most clear and thorough, with a wealth of clear illustrations. The chapter on methods of investigation in relation to the diagnosis, best mode of treatment, and prognosis, is of great value, as is that on the different operations, with a critical account of their value.

Adrenal gland surgery (Broster) and pathology (Vines) represent an interesting phase of new surgical work. To this section is relegated the only coloured plate in the book, which shows the special reaction of normal and abnormal tissue to the fuchsine stain.

A whole section of the book is devoted to what is termed 'Injection Therapy'. This includes the treatment of hæmorrhoids and varicose veins (Harvey), hydrocele and varicocele (Maingot), and chronic gravitational ulcers of the leg. We are surprised that it should be deemed wise or necessary to describe the injectional treatment of varicocele and hydrocele. But our surprise is still greater to find the injectional treatment of hernia seriously advocated by Delisle Gray.

A short chapter on Infections of the Hand is by Hamilton Bailey. The teaching fully adopts Kanavel's work, and is well illustrated by many clear diagrams.

The last section is on 'Orthopædics' and is by St. John Buxton. We think that some explanatory note should introduce this article to explain the curious way in which the subject matter has been chosen. 'Deformities of the feet' include no account of ordinary club-foot (equino-varus); 'disabilities of the knee-joint' make no mention of injuries of the crucial ligaments or of genu valgum or varum; 'acute arthritis' is discussed, but not chronic. Open fractures are 'orthopædic', but not the simple ones; neither deformities of the spine, congenital dislocation of the hip, nor poliomyelitis find any place in this section, and we are left to suppose that they will be dealt with elsewhere.

As we said in reviewing the first volume, the work is of outstanding merit, and a great tribute to the energy and scientific acumen of its many authors. The printing is large and clear, and the illustrations in black and white lavish in the extreme. In fact we think that the number of figures is excessive. It is unnecessary to have ten illustrations of parotid tumours, and many figures from instrument makers' catalogues, e.g., a 'cystoscopic' stool and a stand for a diathermy apparatus, are superfluous. We sincerely trust that the contents of the last volume will allay our apprehensions about the matters of bone and joint surgery.

Minor Surgery and the Treatment of Fractures (Heath: Pollard: Davies). By GWYNNE WILLIAMS, M.S., F.R.C.S., Surgeon, University College Hospital. Twenty-first edition. Crown 8vo. Pp. 485 + viii, with 284 illustrations. 1936. London: J. & A. Churchill Ltd. 10s. 6d. net.

A BOOK which first appeared in 1861, and which has now reached its twenty-first edition, hardly needs any commendation.

We are glad to note, however, that the present edition has acknowledged its first founder, Christopher Heath, by giving a frontispiece portrait and an epitome of his founding this work. The art of bandaging no longer finds a place in the title, but is adequately described and illustrated. Fractures of the long bones and of the spine are well described, and due prominence given to the modern methods of splinting by plaster-of-Paris. We think that an inclusion of Cramer's wire splinting would have been useful. The clearness of the line drawings leaves nothing to be desired.

Demonstrations of Physical Signs in Clinical Surgery. By HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital, London, etc. Fifth edition. Large 8vo. Pp. 287 + xii, with 341 illustrations. 1935. Bristol: John Wright & Sons Ltd. 21s. net.

IN his Ramon Guiteras Lecture delivered in 1932, Henry Wade said, "The wards are the greatest of all research laboratories." This statement is used as a motto for this new edition, and the whole book is a tribute to the value of the clinical work which it enjoins. Nearly all the illustrations are original and most of them are excellent, but it would be an advantage to have still more of the telling diagrams copied from the author's case notes. It seems strange that the book still lacks an adequate illustration of Dupuytren's contracture and of the malignant umbilical nodule.

It is almost unnecessary to say anything in recommendation of this well-known work, though it is a pleasure to record once again that it fully maintains the very high standard of its predecessors.

Urology in Women: A Handbook of Urinary Diseases in the Female Sex. By E. CATHERINE LEWIS, M.S.(Lond.), F.R.C.S., Surgeon to the Royal Free Hospital; Surgeon and Urologist to the South London Hospital for Women. Second edition. Demy octavo. Pp. 100 + viii, with 31 illustrations. 1936. London: Baillière, Tindall & Cox. 6s. net.

THIS book was reviewed in this Journal in 1932, and we congratulate the author on the well-deserved compliment implied by the need for a second edition.

In this new edition sections have been added on obstruction of the vesical neck in women and on endometriosis. In addition, some of the accounts of treatment have been amplified in the light of further experience, and lastly there is a full description of the changes that occur in the ureters during menstruation and pregnancy, which is based largely on the work of Baird.

One of the most interesting sections is that on radium injuries to the bladder; these have been known to ensue on the use of radium for non-malignant affections of the uterus, such as fibroids. The attention of both gynæcologists and radium experts should be directed to preventing these ulcers, which may be the source of danger as well as of great inconvenience to the patient. The author points out that their presence may not be detected until many months after the original treatment; in one case the patient nearly lost her life through hæmorrhage two years after the use of radium for menorrhagia.

No mention is made of the use of mandelic acid in the treatment of chronic bacillus infection, though the author lays emphasis on the difficulty some patients experience in persevering with the ketogenic diet; we should have liked to have read her opinion on this.

One statement is open to criticism; on page 95 she states that hydronephrosis is the commonest complication of nephroptosis; in view of recent research on this condition, we should like to know whether this unqualified statement springs from her own experience.

With the exception of one misprint—on page 42 'tongue' should obviously be 'trigone'—the book is admirably turned out; the illustrations are good, and there is a useful and not too voluminous bibliography.

Diagnostic Roentgenology. Edited by Ross GOLDEN, M.D., Professor of Radiology, the College of Physicians and Surgeons, Columbia University; Director of the Department of Radiology, Presbyterian Hospital, New York. 9½ × 7¼ in. Pp. 867 + xi, with 964 illustrations. 1936. New York and Edinburgh: Thomas Nelson & Sons. $20.00.

THIS is a very complete work on diagnosis by means of X rays. The following quotation from the Editor's Preface gives a good idea of the underlying principle which pervades the work: "The information obtained from an X-ray examination, even when it points clearly to a definite pathologic diagnosis, must be correlated with the history, the physical examination, laboratory findings, etc. This can best be accomplished by the co-operation of clinician and radiologist. The former must not expect his clinical impression always to be confirmed, and must be prepared at times to discard certain preconceived ideas in regard to a case; the latter, while considering the clinical evidence and 'what ought to be there', must at the same time keep an objective and unbiased point of view to avoid overlooking something unsuspected by the clinician. The importance of cordial sympathetic co-operation of clinician and roentgenologist and frank discussion of specific clinical problems cannot be over emphasized."

The book is thus much more than a mere X-ray atlas, although it is indeed a wonderful collection of roentgenographic records. Every section consists of a discussion of the problems of diagnosis and pathology of each organ or system, illustrated by typical radiograms. The skull; the accessory sinuses; the chest wall; the lungs, pleura, bronchi; the heart and great vessels; the stomach, intestines, liver, gall-bladder; the bones, normal and pathological, at all ages; the joints; the spine, spinal cord tumours; the kidneys, ureters, and bladder; the uterus and Fallopian tubes; the bony pelvis and certain aspects of fractures which may cause difficulty in diagnosis: all are treated fully and clearly.

Obviously this book will form an invaluable work of reference for physicians, surgeons, and gynæcologists.

Tumours of the Urinary Bladder. By EDWIN BEER, M.D., F.A.C.S., Visiting Surgeon, Mt. Sinai Hospital; Consulting Surgeon, Bellevue Hospital, New York City. Large 8vo. Pp. 166 + vi, with 52 illustrations (8 in colour). 1925. London: Baillière, Tindall & Cox. 16s. net.

THIS monograph deals with the pathology, the diagnosis, and, more particularly, the treatment of bladder tumours; as such, it is a valuable contribution towards the elucidation of a very obscure and difficult problem of surgery.

The author speaks with authority, for he and his colleagues at the Mount Sinai Hospital have dealt with some hundreds of these tumours, and they have the advantage of a very efficient 'follow-up department'.

Undoubtedly the most important conclusion reached by Dr. Beer is that, in spite of an extensive trial of radium and deep X-ray therapy, treatment of malignant vesical tumours by surgical excision is the most satisfactory.

There are several points of interest in the chapter on the destruction of papillomata by diathermy: no anæsthetic is used in such cases, as pain implies a burn of the bladder wall and thus acts as a danger signal to prevent a traumatic perforation of the viscus; an unsatisfactory result after diathermy should arouse a strong suspicion that the papilloma is malignant; lastly, tumours at the bladder neck are treated by the resectoscope and not by the use of a retrograde cystoscope—indeed, we are surprised to find no mention of Swift Joly's beautiful instrument.

The technique employed in partial resection of the bladder is similar to that used by English surgeons; there is emphasis laid on one point of the operation, and that is that the tumour should be examined from the outer side of the bladder in order to determine the amount of infiltration before deciding on the scope of the excision. The author has found, contrary to the experience of research workers, that there is no marked increase in the bladder capacity following a partial resection; and that oblique implantation of the ureter into the bladder wall is difficult and quite unnecessary, a finding which is confirmed by the observation of kidney function some time after implantation, for intravenous pyelography shows little impairment.

There is an excellent account of total cystectomy, of which no less than eighteen cases are described; of these, five died and six are apparently cured. Dr. Beer advocates bringing the ureters out through the skin close to the anterior superior spines; he is the first to acknowledge that it is an unpleasant business for the patient, but he points out that the mortality of a preliminary implantation into the bowel varies from thirty to fifty per cent.

The illustrations are good, and there is a bibliography; we congratulate the author on a first-class piece of work, and cordially commend the book to our readers.

Die Chirurgie der Hirngeschwülste. By Prof. Dr. N. GULEKE, Direktor der chirurgischen Univ.-Klinik, Jena. Part 5 of *Vorträge aus der praktischen Chirurgie*, edited by Prof. Dr. ERICH LEXER (Munich). Royal 8vo. Pp. 40. 1936. Stuttgart: Ferdinand Enke. RM. 2.20.

THIS small monograph gives Professor Guleke's experience of tumours of the brain and their treatment, over a considerable period. He considers that ventriculography and arteriography allow a local diagnosis in a very high percentage (94 per cent) of tumours. Meningiomas, tumours of the hypophysis, acoustic nerve tumours, and benign gliomas can be removed entirely. For malignant gliomas in elderly subjects, and in medulloblastomas in children, a decompression operation followed by X-ray therapy should be adopted. There seems to be no success with X-ray therapy without operation. A cerebral tumour should be operated upon as soon as the diagnosis is established, in order to avoid such complications as sudden increase of pressure, hæmorrhage into the ventricles, and blindness. Radical operation of the tumour should be done if possible. Partial removal has the advantage of giving more room to the brain and tumour, but malignant change may occur later.

Local anæsthesia should be employed in all operations for tumours of the cerebrum. To avoid fluctuation of pressure during operation, hypertonic solutions of glucose are given intravenously, and magnesium sulphate rectally, as well as puncture of the ventricles. The dura is opened very slowly and the tumour is removed by the electric loop. These operations can be done in one stage, except in cases where there are dangerous fluctuations of blood-pressure. It is possible, nowadays, to remove not only tumours of the ventricles and their neighbourhood, but also cysts situated between the ventricles. Tumours of the hypophysis should be removed transfrontally.

Professor Guleke considers that the prognosis of operations on the brain has greatly improved during the last twenty years. The mortality has decreased from 40–80 per cent to 20–30 per cent, whilst Cushing has a mortality of only 16·6 per cent. In one-third of operated cases a lasting cure has resulted.

The Study of Anatomy. Written for the Medical Student. By S. E. WHITNALL, M.A., M.D., B.Ch.(Oxon.), M.R.C.S., L.R.C.P., F.R.S.(Canada), Professor of Anatomy in the University of Bristol. Third edition. Crown 8vo. Pp. 113. 1936. London: Edward Arnold & Co. 4s. 6d. net.

THIS little book, which costs only four shillings and sixpence, is surely well worth digestion by any medical student. The title is the worst part of it—*The Study of Anatomy*. This makes one think that it is a pocket text-book. It is not the least like that, but rather a brief philosophic talk of a friendly and human kind. Osler would have loved it. It breathes his spirit. Need we say more?

Les petites Règles de la Chirurgie parfaite. By J. OKINCZYC, Professeur agrégé; Chirurgien des Hôpitaux de Paris. Royal 8vo. Pp. 60. 1936. Paris: Masson et Cie. Fr. 12.

THIS pamphlet in French is in the form of a lecture sermon to a new house surgeon. Rather didactic in character, it tells him how to assist at an operation, how to dab, how to tie knots, how to use a Reverdin needle, how to retract, and how to stitch up a wound. Simple notes are laid down for pre- and post-operative care. It is full of useful tips not always found in a text-book, and the style, though colloquial, is occasionally witty.

Gefässmissbildungen und Gefässgeschwülste des Gehirns. By Prof. Dr. HILDING BERGSTRAND (Stockholm); Prof. Dr. HERBERT OLIVECRONA (Stockholm); and Prof. Dr. WILHELM TÖNNIS (Würzburg). Large 8vo. Pp. 181, with 137 illustrations. 1936. Leipzig: Georg Thieme. Paper covers, RM. 24; bound, RM. 26.

VASCULAR tumours of the brain are being discovered more frequently each year in the various neurological clinics both at home and abroad. The present monograph is the outcome of the investigations of clinical and pathological material by Professors Bergstrand, Olivecrona, and Tönnis, in Stockholm. The clinical and pathological material is well set forth, and the illustrations are excellent, there being some 137 of these. The arteriograms are very instructive and clearly show the value of injection therapy in the diagnosis of vascular tumours of the brain. There is an excellent bibliography, which contains nearly 400 references, and is to be found at the end of the monograph. Vascular tumours of the brain represent some 2 per cent of all brain tumours, and in the past must have been missed in most of the clinics.

This monograph forms a useful companion to that published by Harvey Cushing in 1928.

Lehrbuch der Kriegschirurgie. By Dr. CARL FRANZ, Honorarprofessor der Universität, Berlin; etc. Second edition. Large 8vo. Pp. 424 + vii, with 121 illustrations. 1936. Berlin: Julius Springer. Paper covers, RM. 26.60; bound, RM. 28.80.

IT is a matter of some significance that this text-book of military surgery, which was first published in 1919, should now appear in a second edition. The preface to this edition points out that conferences, leagues, and pacts have failed to prevent wars, and that therefore military surgery must be kept up to date.

An excellent feature of the book is the recognition given to methods of treatment used by all the nations engaged in the Great War. The value of the various patterns of Thomas splint both for transport and treatment of fractures is fully recognized and illustrated. It is remarkable to note what a large proportion of space is devoted to the injuries of the limbs, the blood-vessels, and the nerves, whilst the gunshot wounds of the abdomen are compressed into sixteen pages. One is tempted to wonder whether there is any English book ready for publication which would summarize and standardize military surgery in readiness for the next war.

The Human Foot: its Evolution, Physiology and Functional Disorders. By DUDLEY J. MORTON, Associate Professor of Anatomy, College of Physicians and Surgeons, Columbia University. Large 8vo. Pp. 244 + xvi, with 100 illustrations. 1935. New York: Columbia University Press. London: Oxford University Press (Humphrey Milford). 15s. net.

THIS book has been written from an aspect that will be unfamiliar to many who concern themselves with the treatment of foot disorders.

The first part of the book, devoted to the evolutionary development of the foot, consists of a critical survey of the problem of the terrestrial locomotion of living organisms, and the stages which it is assumed have led to the development of the arched human foot as we know it. Though chiefly concerned with the skeletal changes, the author deals also briefly with the muscles.

The second part deals with the physiology of the foot both of civilized and primitive communities. The author considers the foot with regard to its function of balance and weight supporting, and later as an agent of locomotion. It will be found that he gives greater prominence to the part played by the ligaments in arch support than will be accepted by all authorities, and produces interesting evidence in criticism of the widely accepted conception of an anterior transverse arch.

The third part of the book describes certain functional disorders which have come to the author's notice. As the preceding chapters show that the arched foot is a human characteristic which has been acquired slowly and laboriously throughout the development of the race, it is not surprising that these functional defects concern imperfections of this arch structure.

His analysis is based on data derived from clinical investigation, X-ray evidence, and records made by his staticometer and kinetograph. The main defects he finds are: short first metatarsal bone, posteriorly placed sesamoid, and hypermobility of the first metatarsal segment. He concludes by considering the principles of treatment and the methods which can be used to produce correction of weight distribution.

The book is based on the results of careful original investigation and will be of great value to anatomists and orthopædic surgeons.

Hero-Dust. By JAMES KEMBLE, Ch.M., F.R.C.S. Pp. 193 + xvi, 1 illustration. 1936. London: Methuen & Co. Ltd. 6s. net.

MR. JAMES KEMBLE, the author of *Idols and Invalids*, now contributes an equally readable volume which he calls *Hero-Dust*. It is written primarily for laymen, but contains much that will give thought to medical men, for Mr. Kemble has read widely and is able to throw light upon his problems by his knowledge of medicine and surgery. The longest and perhaps the most important essay deals with the blindness of John Milton; from the details given it is clear that it was due to chronic glaucoma attacking first his left, and some three years later his right, eye. Dealing with Catherine the Great, the author tells the story of her inoculation with small-pox by Dr. Dimsdale, one of whose descendants became Lord Mayor of London. He tries also to unravel the paternity of her children, but without complete success, as she is known to have had twelve lovers from the time when she was 23 until she reached the age of 60. The troubles of Mary Queen of Scots he considers from a new angle; he believes that she was with child by Bothwell before she married him, and that she was six months' pregnant when she miscarried. Beau Brummell and Omar Khayyám conclude a little volume which will pass away a leisure hour with pleasure and profit.

My Life and Work: The Search for a Missing Glove. By Dr. ADOLF LORENZ, Hofrath and Professor of Orthopædic Surgery, University of Vienna. Demy 8vo. Pp. 362 + xi. Illustrated. 1936. New York and London: Charles Scribner's Sons Ltd. 12s. 6d. net.

DR. ADOLF LORENZ tells in this autobiography how from nothing he rose to be Professor of Orthopædic Surgery in the University of Vienna and was complimented with the title of Hofrath. He tells the story modestly and well, speaking of his early struggles, of the reasons which led him to adopt orthopædic surgery, and of his success, more especially in the treatment of congenital displacement of the hip. Many will still remember the demonstrations he gave at Liverpool when on his way to visit the United States for the first time, and of the number of imitators he had in this country. But he says comparatively little of the surgical side of his work. The book is chiefly personal, and as such is extremely good reading, for it is the life history of one who became, as he always intended to be from his infancy, 'ein grosser Herr'. From the depths of poverty he attained to wealth, and from riches he sank again into poverty, as Austria was ruined by the war and he had invested his savings in home securities. Nothing daunted, he set to work again in the United States, and by his appeals was able to send help to the many children whom he knew were starving in Vienna. Prof. Lorenz paints himself as an optimist, somewhat of a mystic with an inherent dread of snakes, a gourmet always longing for his native dishes, with a peasant's hunger for land and yet with a love for building and pretty surroundings. He has been fortunate in securing a wife who acted for many years as his assistant in practice, who lives happily with him, and curbs his tendency to extravagance. There is an excellent index and the illustrations are well reproduced.

BOOK NOTICES

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.*]

The Operations of Surgery. By R. P. ROWLANDS, M.S. (Lond.), F.R.C.S., late Surgeon to Guy's Hospital, etc.; and PHILIP TURNER, B.Sc., M.S. (Lond.), F.R.C.S., Consulting Surgeon to Guy's Hospital, etc. Eighth edition. Volume I: The Upper Extremity. The Head and Neck. The Thorax. The Lower Extremity. The Vertebral Column. Royal 8vo. Pp. 1405 + x, with 435 illustrations (38 in colour). 1936. London: J. & A. Churchill Ltd. 36s. net.

Manual of Emergencies: Medical, Surgical and Obstetric: Their Pathology, Diagnosis and Treatment. By J. SNOWMAN, M.D., M.R.C.P.Lond. Third edition. Crown 8vo. Pp. 401 + ix. 1936. London: John Bale, Sons & Danielsson Ltd. 10s. 6d. net.

Treatment of Fractures in General Practice. By W. H. OGILVIE, M.D., M.Ch., F.R.C.S. Second edition. F'cap 8vo. In two volumes. Pp. 180 + viii, with 37 illustrations. 1936. London: John Bale, Sons & Danielsson Ltd. 2s. 6d. each net.

Die Nachbehandlung nach Operationen: ein Lehrbuch in Vorlesungen. By Prof. Dr. PAUL REICHEL, Geheimer Sanitätsrat. Third edition. Large 8vo. Pp. 499+xii, with 85 illustrations. 1936. Munich: J. F. Bergmann. Paper covers, RM. 24; bound, RM. 25.80.

Roentgenographic Technique: A Manual for Physicians, Students, and Technicians. By DARMON ARTELLE RHINEHART, A.M., M.D., F.A.C.R., Professor of Roentgenology and Applied Anatomy, School of Medicine, University of Arkansas, etc. Second edition. Large 8vo. Pp. 431, with 183 illustrations. 1936. London: Henry Kimpton. 28s. net.

Cystoscopy and Urography. By JAS. B. MACALPINE, F.R.C.S., Hon. Surgeon and Surgeon in Charge of the Genito-urinary Department, Salford Royal Hospital, Manchester. Second edition. Large 8vo. Pp. 478 + xv. with 297 illustrations and 14 coloured plates. Bristol: John Wright & Sons Ltd. 30s. net.

Orthopædic Surgery. By WALTER MERCER, M.B., Ch.B., F.R.C.S.(Edin.), F.R.S.(Edin.), Assistant Surgeon, Royal Infirmary, Edinburgh, etc. With a Foreword by JOHN FRASER, *M.C.*, M.D., Ch.M., F.R.C.S.E., Regius Professor of Clinical Surgery in the University of Edinburgh. Second edition. Medium 8vo. Pp. 906 + xi, with 408 illustrations. 1936. London: Edward Arnold & Co. 40s. net.

A Handbook of Urology for Students and Practitioners. By VERNON PENNELL, M.A., M.B., B.Chir. (Cantab.), F.R.C.S. (Eng.), Hon. Surgeon and Surgeon with Charge of Urological Department, Addenbrooke's Hospital, Cambridge; etc. Cr. 8vo. Pp. 224 + viii, with 34 illustrations. 1936. London: Cambridge University Press. 7s. 6d.

Pathology of the Nervous System. A Student's Introduction. By J. HENRY BIGGART, M.D. (Belfast), Pathologist to the Scottish Asylums' Board; Neuropathologist to the Royal Infirmary, Edinburgh; etc. With a Foreword by Professor A. Murray Drennan, M.D., F.R.C.P. Demy 8vo. Pp. 335 + xvi, with 204 illustrations. 1936. Edinburgh: E. & S. Livingstone. 15s.

A Descriptive Atlas of Radiographs. An Aid to Modern Clinical Methods. By A. P. BERTWISTLE, M.B., Ch.B., F.R.C.S. (Edin.). Third Edition, revised and enlarged. Cr. 4to. Pp. 560 + xxxi, with Frontispiece and 794 illustrations. 1936. London: Henry Kimpton. 42s.

La Pratique Chirurgicale Illustrée. Fascicule XX. Large 8vo. Pp. 259, with 219 illustrations. 1936. Paris: G. Doin et Cie. Fr. 70.

Le Cancer (Etudes Anatomo-Cliniques).—By I. STOIA, Docent de la Faculté du Médecine de Bucarest; Médecin chef du Laboratoire central d'Anatomie-Pathologique de l'Hôpital Brancovan; etc.; and P. STANCIULESCU, Assistant du Laboratoire central d'Anatomie-Pathologique de l'Hôpital Brancovan; etc. With a Preface by Professor CH. LENORMANT. Super royal 8vo. Pp. 322, with 147 illustrations. Paris: Masson et Cie. Fr. 55.

Fasciæ of the Human Body and Their Relations to the Organs They Envelop. By EDWARD SINGER, M.D., Department of Anatomy, College of Physicians and Surgeons, Columbia University. $10\frac{3}{4} \times 7\frac{3}{4}$. Pp. ix + 105, with 24 illustrations. 1935. Baltimore: The Williams & Wilkins Company. (London: Baillière, Tindall & Cox.) 13s. 6d. net.

Studies on the Healing of Fractures with Special Reference to the Significance of the Vitamin Content of the Diet. By JOHN HERTZ, M.D., Assistant Surgeon, Department C, Surgical University Hospital, Copenhagen. Super royal 8vo. Pp. 286, with 82 illustrations. 1936. Copenhagen: Levin & Munksgaard. (London: Oxford University Press.) Kr. 15.

Roentgendiagnostik der Knochen- und Gelenkkrankheiten. By Professor Dr. ROBERT KIENBÖCK (Vienna). Part 4. Degenerative Wirbelsäulenerkrankungen. Large 8vo. Pp. 229–450, with 214 illustrations. 1936. Berlin and Vienna: Urban & Schwarzenberg. RM. 24.

A Short Practice of Surgery. By HAMILTON BAILEY, F.R.C.S. (Eng.), Surgeon, Royal Northern Hospital, London, etc.; and R. J. MCNEILL LOVE, M.S. (Lond.), F.R.C.S. (Eng.), Surgeon, Royal Northern and Metropolitan Hospitals, etc. Third edition. Demy 8vo. Pp. 995 + viii, with 996 illustrations, of which 88 are coloured. 1936. London: H. K. Lewis & Co. Ltd. 28s. net.

In Memoriam

LORD MOYNIHAN

1865-1936

In Memoriam

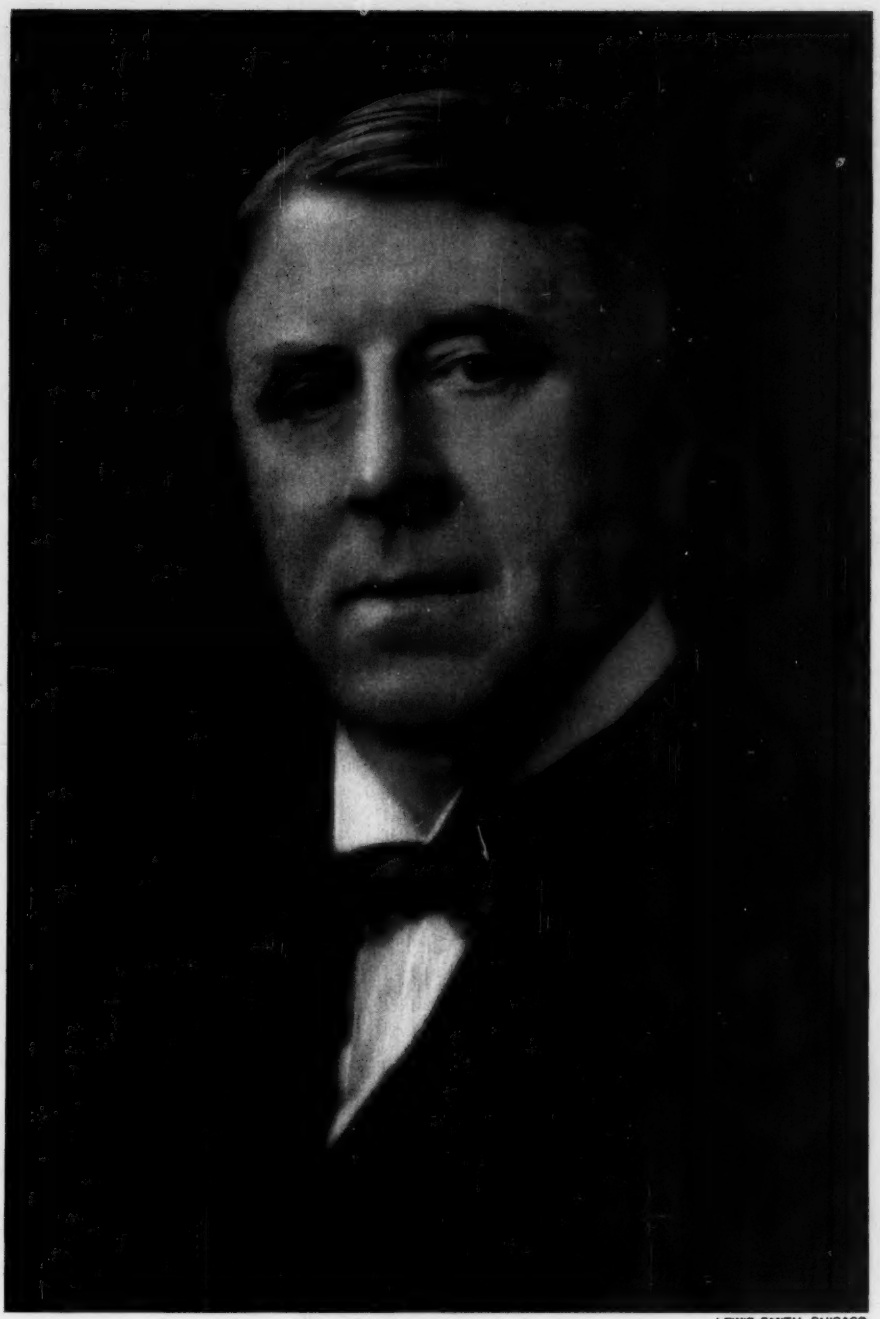
MOYNIHAN

It is difficult to believe that Moynihan, that vivid, compelling personality, has really gone. It is a happy thought that his teaching and his work live on, to remind us of what can be accomplished in one lifetime by a man of single purpose and unswerving devotion to a great ideal.

He himself often referred to the Editorial Committee of this Journal in Nelson's words as " A band of brothers ". Those of us who composed that Committee worked together for twenty-three years in unbroken harmony. In the early days of anxiety, when our enterprise was young and struggling for existence, we used to look forward to the remote possibility of coming of age, and when we had attained the mature age of 21 years it was felt that some tribute should be made to our Chairman. We hesitated as to whether we should wait until we were a quarter of a century old, but by a happy chance we decided this year to present our testimonial without further delay. That presentation, together with two portraits of Moynihan in his youth and of Moynihan clothed in the splendid robes which marked his highest achievement, was described in our last issue. Little did we think then that ' Ave ' was also to be ' Vale '. No man ever had a fuller life, or one more richly rewarded with success.

Of untiring energy of body and mind, he climbed from step to step up the ladder of fame and fortune. Every cause which he took up gained strength and lustre from his leadership.

The Leeds Medical School, the British Journal of Surgery, the Council of Army Consultants, the Royal College of Surgeons, and most of all the science and art of abdominal surgery, all felt the magic of his touch. His perfect technique, combining gentleness with daring, made him at once a master and a teacher of his art.



LEWIS SMITH, CHICAGO

LORD MOYNIHAN

1865-1936

CHAIRMAN OF THE EDITORIAL COMMITTEE

1913-1936

UNIV. OF MICH.

He elucidated the early diagnosis of duodenal ulcer and of diseases of the biliary tract; he added to our knowledge of diseases of the spleen and of the pancreas. He discovered and taught the science of "the pathology of the living".

He brought surgeons of the provinces together, he associated the provincial with the metropolitan surgeons in the pursuit of those ideals which he himself had followed.

His gift of lucid and persuasive oratory enabled him to sway men's minds and emotions.

To those of us who have been privileged to be closely associated with him it will seem for the rest of our lives that "he being dead yet speaketh".

UNIV. OF MICH.